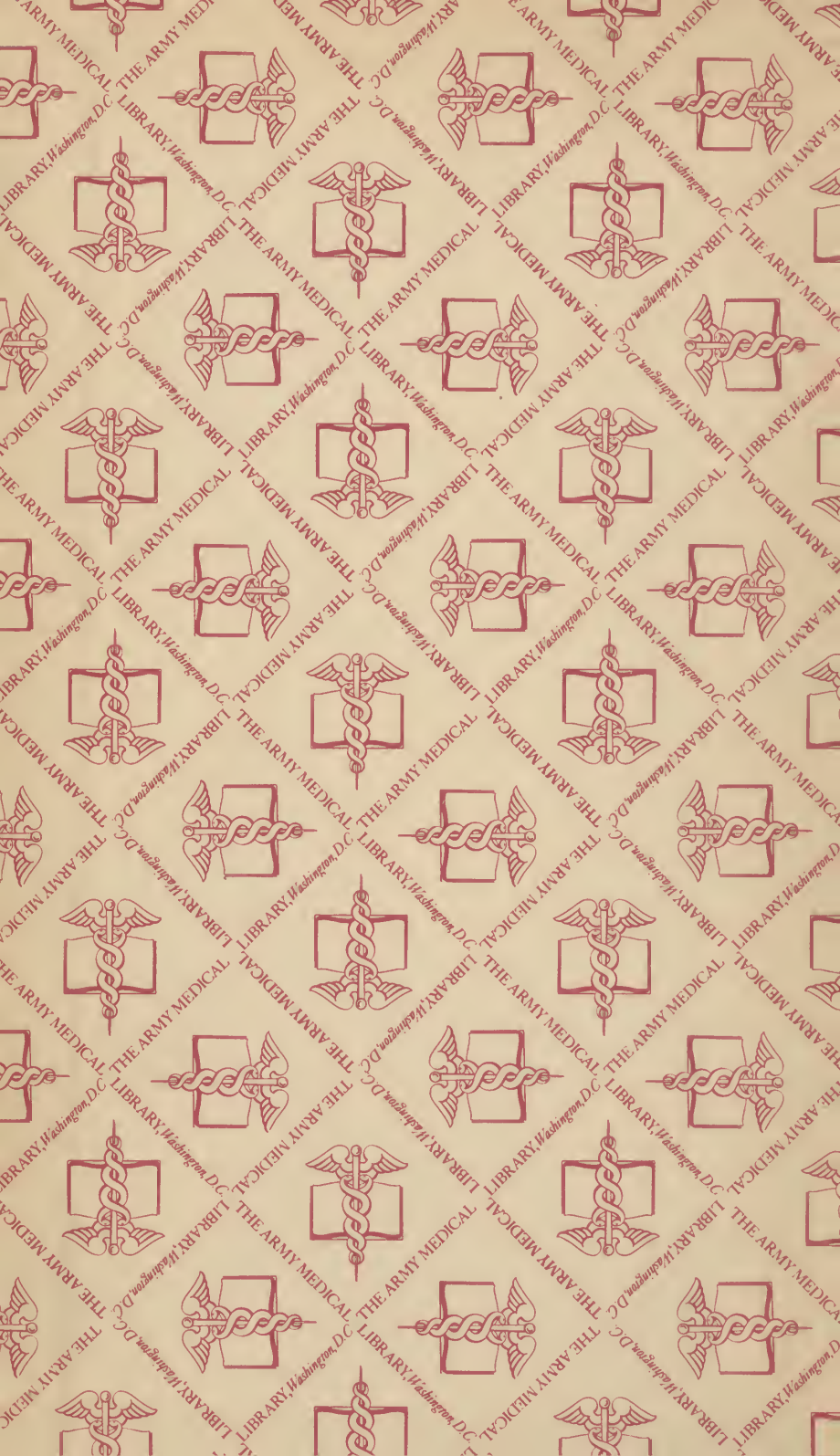
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Gentlemen:—The eruptive fevers are the most typical examples of specific diseases. Before proceeding any farther in the study of the cases which we are observing together, I wish to pause for a little to examine the subject of specific character in disease. I hope to be able to show you that this important question is dominant throughout the whole of pathology and therapeutics—in fact, throughout the whole of medicine. I have already, in former lectures, had opportunities of bringing this subject under your notice. In practice, you will find it confronting you at every step; and as not a day will pass without your hearing me refer to it at the bedsides of the patients, I must endeavour to give you as complete an idea as possible of that which is understood by the term *specific*, when applied to diseases.

Though we are in the habit of saying that we have emancipated ourselves from the yoke imposed by the doctrines of Brown and of Broussais, we really are still under their influence: although we repudiate them, they are recchoed in our medical speculations, and in the very language we employ. It, therefore, becomes necessary for me to recall to your recollection the errors which are embraced in these doctrines. However much the two doctrines may be opposed to each other, they rest on a common basis; for although Broussais was the great antagonist of Brown, he nevertheless de-

rived the principles of his physiology from the pathological system of the Scottish reformer, whose *incitability* differed in an abstract manner only from the *irritability* of Broussais.

Brown said that life was maintained by *incitants:* Broussais said that it was maintained by *stimulants.*

Their physiological theory was founded on this assumption; and on it likewise was based their pathological hypothesis. In point of fact, they both held that there was only one morbid cause, the excessive or unreasonable use of incitants or stimulants. Difference in the intensity of the cause, and difference in the mode of the reaction of the economy are, they said, the sources of the innumerable diversities of form presented by diseases. This is their common starting point; for here "incitants" and "stimulants" are two absolutely synonymous words.

Brown said,[1] and Broussais repeated in other terms, that light is the natural incitant, or, what comes to the same thing, the natural stimulant of the eye, the incitation of that organ being vision; that food is the natural incitant of the stomach, the result of the incitation of which is digestion; that the assimilated materials, the nutritive juices, are the natural incitants of different organs, whence we have nutrition; that the blood is the natural incitant of the organs of secretion, whence, for example, when the kidneys are concerned, we have the urinary secretion, and when the seminal glands are concerned, the spermatic secretion. While Brown and Broussais held that the cause was always the same, varying only in degree, they could not avoid admitting the existence of a modifying influence in the structure of individual organs, in virtue of which the effects of stimulation are different. Their assertion that every thing was dependent upon the quantity of the stimulus, and that there was an identity in the condition of organs in all persons, was a denial of evident facts. Upon their hypothesis, how can diversity of effects, that is diversity of functions, be explained? Does not their hypothesis involve prodigious absurdities, absurdities quite as great as that which Récamier, a man of undoubted talent, was led into—to the effect, that by exalting the incitability of the nerves of the finger or epigastric region, to a degree of incitability equal to that of the retina, we could see with the finger or the stomach, on adapting to them an optical apparatus similar to the eye?

---

[1] Brown, (John, M.D.):—Elements of Medicine: London, 1795.

Brown and Broussais, then, were obliged to admit that there exists diversity in the manifestations of vital power due to the special anatomical properties of tissues and organs, of solids and liquids, as well as diversity in the functions therewith connected: but they did not take them into account. The fundamental idea of their doctrines is identical: this Broussais has recognised by adopting as the test of his own doctrine the synthetic proposition of Brown, that all diseases are sthenic or asthenic, that is, dependent either upon excess or deficiency of excitement; but by the manner in which he interprets the effects of reaction, he completely diverges from the path of his predccessor, and arrives at therapeutical conclusions entirely opposed to those of the disciple of Cullen.

Brown maintained that all parts of the body are endowed with a particular special aptitude which he called *incitability*. This incitability, he said was manifested by incitation, and this incitation could only be the result of an inciting force: but this aptitude is limited. Since whenever it is brought into play, it becomes by that very circumstance exhausted, so it requires to be constantly renewed by augmentation of the quantity of the force by nutrition; or by an accumulation of force taking place through non-expenditure consequent upon repose of the organism. Thus, by movement, the incitability of muscles is exhausted, and when muscular action has been excessive in degree or too prolonged, the individual, being in the last stage of fatigue, loses the power of moving. Thus, Gentlemen, you perceive how both the pathological and therapeutical doctrine of Brown wholly originates in this fundamental fact.

According to Brown, every disease depends either upon diminished incitability, the consequence of excessive incitation, or an increased incitability the effect of a diminution of incitation. In both cases, the final result is debility; and consequently, the part of the physician ought always to be restricted, first, to the restoration of the vital powers by very moderate stimulants, and in the second place, to the use of means capable of augmenting the incitability.

Broussais, taking an isolated view of the irritability which exists in tissues, held that all diseases spring from the untimely or excessive action of agents having the power of exciting that irritability. According to his view, therefore, the only morbific causes are irritants; and the effects which they produce is irritation. Holding the opposite of Brown's opinion, he thought that it was necessary

to restore the functions to their physiological condition, and to endeavour to calm and remove the irritation.

Whether the pathological state consists, according to the Edinburgh doctrine, in a greater or less degree of incitability, or according to the Val-de-Grâce theory in an excess or more rarely in diminished irritability, in these dichotomic symptoms, (essentially opposed to each other, though having one and the same origin,) the quantity only, and in no way the quality, of the morbific cause is taken into account. Treatment of disease based on such systems must necessarily possess extreme simplicity. Brown confined himself to the use of the class of remedies known as excitants, using in some very rare cases antisthenics, if I may be allowed to use such a term; while Broussais always employed antiphlogistic medicines, except when, under very exceptional circumstances, he prescribed excitants.

There is no denying that a certain class of acute inflammations are pretty exactly comprehended within the description applicable to the system of Broussais; for that which renders an inflammatory disease more or less serious is, on the one hand, the greater or less intensity of the cause under the influence of which it has been developed, and on the other, the difference of the organizations which are affected. But there is another class of diseases which has not this dichotomy: it is the class of specific diseases. It mattered very little to Brown whether small-pox was a specific disease. All he required was to ascertain whether the malady was sthenic or asthenic, to enable him to formulate the therapeutic indication of stimulating or lowering. It mattered very little to Broussais whether cholera differed in form from dothinenteria; in both diseases, he saw irritation of the digestive canal setting up different symptoms, but the irritation was to him the dominant fact which constituted the necessity for antiphlogistic treatment.

Thus it was, that the whole of nosology and therapeutics became a *tabula rasa*. Matters were in this state at the beginning of the century. The doctrine of Broussais, on first acquaintance seductive from its simplicity, had obtained many adherents when Laennec and Bretonneau, each from his own point of attack, dealt a blow at it, the formidable character of which Broussais tried in vain to conceal. Laennec, under the modest title of a semiological discovery, and seeming to confine his observations to the study of the diseases of the respiratory organs, wrote a very striking chapter on nosology.

While Bretonneau was restoring the history of acute, Laennec restored the history of chronic diseases.

The illustrious physician of Tours overthrew to its very foundations the great edifice of *physiologism* and pretended *rationalism* in therapeutics, and on its ruins raised the doctrine of the existence of a specific element in disease. This he did, by calling attention to the elementary fact, that differences in the nature of the cause impart to diseases much greater differences than those which they derive from greater or less intensity of the cause, or from diversity of organization.[1]

In physiology, Bretonneau attributed to the special properties of different tissues and different organs a much greater importance than that which he accords to the agencies which modify the organism: in pathology, he admitted that a great number of diseases have a common element generally called irritation or inflammation; but he did not accord to this common element the importance assigned to it by Broussais. Undoubtedly furunculus, malignant pustule, syphilitic chancre, herpes preputialis, gastric disturbance and dothinenteria have, as an element in common, inflammation characterised by fluxion, by redness appreciable when the inflamed parts are within view, by pain, and by increased temperature; but along with this common element, there are other elements of great importance which distinguish the different affections from each other, and have a significance altogether peculiar.

The natural history of disease has some remarkable analogies with the natural history of plants and animals. This truth was long ago enunciated by Sydenham, when, in one of the chapters of the second section of his *Medical Observations*, he says, in speaking of pestilential fever and the plague of armies which committed ravages in 1665—1666:—"unaquæque morborum non minus quam animalium aut vegetabilium species, affectiones sibi proprias perpetuas ac pariter univocas ab essentia sua promanantes sortita est." Examples in illustration taken from Botany and Zoology will facilitate the understanding of the subject of which I am now treating.

Different kinds of vegetables, for instance, present us with characters in common, in virtue of which we place them in the same natural families; and these common characters are also found in

[1] Bretonneau:—Recherches sur l'Inflammation Spéciale du Tissu Muqueux et en particulier sur la Diphthérite. Paris: 1826.

some proximate families; but in the form of the flower, in the shape of the fruit, in the juices secreted by the plant, there are distinctions which not only prevent families being mistaken for one another, but also prevent the most similar species from being confounded. Thus, dulcamara, datura stramonium, celandine, poppy, the wild briar, and cherry-laurel possess characters in common, but they have each well marked special characters which make it impossible for the botanist to mistake any one of them for the other.

When you study two individuals of the class *reptilia* and the order *ophidia*, the ring-snake and the viper, you notice similitudes in their external forms and anatomical structure, but you bestow special attention upon their specific distinctions. The presence or absence of scales or plates on the head of the animal, the presence or absence of poison-fangs, establish for you fundamental differences between two individuals similar in their general appearance; and you could not commit such an error as to regard the viper as a variety of the ring-snake.

Well, then, Gentlemen, in diseases which seem to bear the strongest resemblance to one another, there are specific characters quite as distinctive as those which distinguish the different species of the same family of plants, or the different species of the same class of animals. Now this is what Broussais was not inclined to admit. The inflammation-element, the existence of which we cannot deny, was, to his mind, the chief and indeed the only dominant fact. It is so in some cases: in a purely inflammatory disease, the quantity of the morbific cause is everything, provided allowance be made for diversity of organs and variety of organization: generally speaking, in the phlegmasiæ, as in the pyrexiæ, as well as in the majority of diseases, we have to consider the quantity less than the quality of the morbific cause.

Let me use as my illustrations facts which admit of being stated with absolute precision, and are consequently facts the least likely to be disputed.

A small vesicle appearing at the base of the *glans penis* after impure intercourse is certainly in appearance a thing of trivial importance: if judged only by its appearance, it is a much less serious affection than a group of vesicles of herpes preputialis, which is liable to appear under similar circumstances. In fact, if the inflammation-element only be taken into account, the herpetic is the more formidable of the two affections. But how vast the differ-

ence, when we proceed beyond that element which they have in common! While the herpetic vesicle, if left to itself, becomes filled with pus, dries up, and leaves in its place, after the formation and fall of a crust, an insignificant cicatrix, the syphilitic vesicle runs, rapidly perhaps, through its stages; but in the place it occupied, you will soon observe an induration of the subjacent cellular tissue, which you will at once recognise as establishing a distinction between the syphilitic and non-syphilitic affection—a distinction which you will consider as of the very highest importance. In this you will assuredly be right; for after recovery from the herpes, you need have no anxiety regarding your patient: when the local malady has disappeared, a radical cure has taken place. Is it so after the cicatrisation of the chancre? No! for after two or three months, and sometimes after a longer interval, manifestations will appear on the skin and mucous membranes, which you will connect with the previous existence of the little insignificant looking vesicle. You will see appear a special cutaneous eruption and ulcerations of the throat; or, if energetic medical treatment does not prevent it, you will have the successive development of other affections originating in the first—affections of the cellular tissue, tubercles on the skin, condylomata, diseases of the bones, such as ostalgia, caries, and necrosis—which if not checked in their progress will lead to terrible disorders. In addition, therefore, to the characters which the syphilitic chancre had in common with the herpetic vesicle, it had specific characters deserving great consideration.

The clinical study of diseases furnishes innumerable examples similar to that now adduced: what I have said of syphilitic chancre, I might repeat in substance of a host of other affections.

A small pimple forms on the hand of a butcher who has been skinning a sheep which has died of malignant pustule [*sang de rate*]. It occasions only a disagreeable feeling of tickling in the part: in comparison with a boil, which is often exceedingly painful, it appears to be scarcely deserving of notice. But by-and-by this pimple so insignificant and apparently so mild in its character, begins to enlarge: a small slough forms in the situation which was occupied by the pimple: an erysipelato-œdematous tumefaction, commencing in the affected part, gradually invades the whole member: the lymphatic glands of the arm and axilla become engorged: at the same time, fever sets in, which increases from day to day: then delirium supervenes: and at last, sooner or later, the individual dies, in a

state of extreme debility, with aggravated typhoid symptoms. The little pimple was a malignant pustule. The boil, on the other hand, which almost from the the very first caused violent pain, and in which the inflammation-element attained a much more intense degree than in the case of the malignant pustule, gets well spontaneously: the patient, who has suffered a great deal of pain has nothing to fear. The inflammation, therefore, did not play a very important part: the quantity of that element was of no consequence—its quality was everything.

The characters which put their special seal on specific diseases are univocal and constantly met with, irrespective of the degree in which exists the common element with which they are associated. Thus, small-pox, whether distinct or confluent, benignant or malignant, normal or modified, will always be found to have its pustules, its own special pustules, which constitute its character, a character as essentially invariable and as specific as the distinctive marks of the natural families of plants, or the natural division of the classes of animals.

That which is true in human, is equally true in comparative pathology. Thus, you will see tag-sore [*clavelée*] that eruptive disease of sheep regarding which I spoke in a previous lecture [vol. II, p. 89], comparing it with small-pox in the human subject, show itself by an eruption presenting characters perfectly precise and univocal, which enables it to be distinguished from all the other eruptive diseases of sheep.

Even plants, so much inferior to animals in the scale of organization, exhibit in their pathological disorders the influence of the quality of the cause, by the form of the disease. The insects which wound the leaves or stems of plants excite at the point of contact the growth of morbid excrescences, the univocal character of which points out the agent by which the wound was made. On plants the same kind of scurf always follows the wound inflicted by the same kind of insect; and this occurs with such constancy, that the experienced naturalist can always determine from the form, colour, and volume of the excrescence the kind of insect to which the contained larvæ belong.

Exactly the same thing occurs in respect of an internal or external inflammation of the human subject. In dothinenteria, there is, in addition to the characters possessed by it in common with all other intestinal phlegmasiæ, an inflammation occupying a circumscribed

locality—a locality which is limited, determinate, and always the same: there is the furuncular eruption of the agminate and solitary glands, and as this furuncular eruption is always found in dothinenteria, it is very properly regarded as the specific character, the special anatomical manifestation of the disease.

In dysentery, which is in reality a form of colitis, special characters are also found: they exist in the intestinal secretions, in the symptoms, and in the anatomical lesions, enabling us to distinguish this form of inflammation of the large intestine from other species of colitis, and to establish the specific character of the disease.

Let me remark, Gentlemen, that the specific characters of which I have been speaking must not be confounded with the characters which constitute mere varieties: in nosology, as well as in natural history, it is necessary to keep the two distinct from one another.

To continue my comparisons: there is a great difference between the lady's pocket-dog and the large dog [*molosse*] of the Pyrenees: still, the two do not belong to different species, but are merely varieties of the same species of the genus *canis*. The same instincts, the same anatomical and physiological characters are invariably found present in both. Ingenious breeders, by skilful crossing, can produce animals very different from the parent stock, can produce breeds in which the wool, the fat, or the muscle predominates according to the use for which the animal is destined; yet these different breeds are merely varieties of one type, all of which preserve the typical specific characters. So it is also in plants: you know how completely we have it in our power to multiply varieties of a vegetable species, and, so to speak, to create monstrosities. Thus, from the simple pink, a skilful horticulturist produces innumerable varieties, just as from the wild briar he obtains the beautiful roses which adorn our gardens.

But both in the animal and vegetable kingdoms, we only produce varieties—different forms of the same species—and we have no power completely to change the characters of species, far less to create new species. It is long since the horse and the ass have been crossed: stallions have been coupled with female asses, and male asses have been coupled with mares: mules have been the only resulting progeny—that is to say, varieties partaking of the characters of both species of the genus *equus*, accidental varieties, however, which are not reproduced, and which do not perpetuate themselves.

In nosology, no more than in natural history, ought variety of type to be taken for difference in species. Varioloid, or modified small-pox, is not a different species of small-pox, but merely a modification or variety: varicella or chicken-pox is an entirely distinct species.

Gentlemen, I insist on this point because some are disposed to see in the specific character of diseases only a question of more or less, while in reality, there is as absolute a difference between different species in nosology as in botany or zoology. Do what you will, you will never succeed in transforming roseola into measles, chicken-pox into small-pox, or simple catarrhal bronchitis into whooping-cough. All these diseases have their absolute and invariable specific character sharply distinguishing them from one another, whatever may be the degree of their severity. The existence of specific character is so indisputable, and is every where so clearly indicated, that it is not necessary for the recognition of a nosological species to have before us an assemblage of all the symptoms: as is seen in "defaced scarlatina" [*scarlatine fruste*], a single word will often be sufficient to enable the physician to recompose the entire pathological phrase, just as Cuvier recomposed lost species of animals by the study of a few fragments of antediluvian skeletons.

Specific diseases derive their invariable characters not from the quantity, but from the quality of the morbific cause: this is invariable in its nature, whatever may be the influence under which it is developed.

The class of special diseases is so vast as to fill the greater part of the nosological table. If we study the different causes of diseases, whether these causes are irritants, or agents of an entirely different kind, we shall see results produced which are so invariably characterised by the same forms, according to the nature of the causes, that it will be impossible not to recognise the specific element at every step as we proceed in our observation of patients.

A blister on the skin differs in its character according to the cause in which it originates; according, for example, to its being the result of the application of cantharides, of a sun-burn, of erysipelas, or of cauterization with ammonia. You know how smarting is the pain of a sun-burn: it is not the same kind of pain which is caused by blistering with cantharides or ammonia—it is more acute and lasts longer; but nevertheless, the cutaneous inflammation caused by blistering with either substance is much more intense than that

which results from a burn by the sun: each cause produces its own special effect.

Let me illustrate the subject by facts still simpler—by the effects of chemical agents, which are very easily ascertained. Each of these agents, when applied to the human body, has, according to its individual nature, its own special action. The pain produced by hydrochloric acid is much more transient than that occasioned by nitric acid; and nitric acid, although it occasions sloughing of the parts which it touches, causes less acute and less persistent pain than cauterization with sulphuric acid, although the destruction of tissues may be less extensive. Every medical student knows that the application of potassa fusa and the alcaline caustics is much less painful than the application of the chloride of zinc, chloride of antimony, or arsenical preparations. To sum up in a few words all that there is to say on the subject:—the different chemical agents produce so distinctive an action that even a person of little experience can declare the substance which has been used from observation of the effects produced. In these cases, one cannot argue from the quantity of the cause, for experience has shown that it is impossible to produce similar effects with caustic potash and chloride of antimony, whatever proportions of these agents may be employed. That this depends on their respective chemical properties, and on the manner in which the agents combine with the tissues, I admit; but that does not signify, if there be a difference, and if that difference be constant.

Let us now examine the action of poisons. All poisons have their own peculiar mode of action, and so characteristic is it, that nearly always the slightest examination of the symptoms suffices to determine the nature of the poison. There is certainly no one at all acquainted with toxicology who cannot distinguish by the symptoms poisoning by opium from poisoning by stramonium, veratria, or strychnia; or who is unable to discriminate the differences in the consequences of absorption of the venom of the rattlesnake, the viper, the scorpion, the tarentula, the bee, or the mad dog.

To every specific morbid cause, the organism responds by the manifestation of effects having a specific character.

A man comes into hospital with paralysis of the extensor muscles: the edges of his gums have a bluish fringe: his skin has a somewhat jaundiced hue: he complains of violent colic, and of shooting pains in the course of the nerves of the limbs: in such a case a

prolonged examination is not required to diagnose poisoning by lead. The nature of the case is so palpable, that one cannot entertain the idea of its being the subject of any doubt. The disease has characters so essentially specific, that it is recognised at a glance, just as a tree is recognised at first sight by its leaves and general appearance. You can at once lay hold of the distinctive characters of poisoning with lead and copper, just as you can distinguish at the first glance the different species of the animal and vegetable kingdom.

Another patient comes into hospital affected with general tremors: his gums are ulcerated and bleeding, and the teeth shake in their sockets: his mind is enfeebled. Our first question is asked to ascertain whether he is a looking-glass manufacturer, a gilder on metals, or engaged in any other occupation in which mercury is employed: we have at once suspected mercurial poisoning. There was, in fact, something so characteristic in the symptoms that we could not mistake them.

You are acquainted, Gentlemen, with the symptoms which characterise the disease produced in the workers in vulcanised caoutchouc factories by inhaling the vapours of sulphuret of carbon. The interesting inquiry of my colleague Dr. Delpech has recently directed attention to this species of poisoning.[1]

This sagacious observer, by marking with care the specific character of the phenomena in the case of a workman in a caoutchouc factory, phenomena which could not be attributed to any known disease, was able to establish the existence of a new disease, of which he has since met with a certain number of cases, all of them presenting the same characteristic symptoms. The symptoms of this new disease are—disturbance of the mental faculties, particularly loss of memory; headache, which is more or less acute, and is sometimes very intense; vertigo, occasionally in an extreme degree; pains in the limbs, accompanied by a sensation of general formication and analgesia, and, in very exceptional cases, with cutaneous hyperæsthesia; impaired power of the organs of sense and reproduction: disturbance of the motor powers, characterised at first by

[1] Delpech, (A.):—Mémoire sur les Accidents que développe chez les Ouvriers en Caouchouc l'inhalation du Sulfure de Carbone en vapeur. Paris, 1856.

Nouvelles Recherches sur l'Intoxication Spéciale que détermine le Sulfure de Carbone. [*Annales d'Hygiène Publique*, 1863: *2e Série*, t. xix.]

cramps and then by muscular contractions; muscular debility, appearing first in the inferior and then in the superior extremities; finally, anorexia and vomiting. Under the influence of these symptoms, the patient falls into a state of more or less profound cachexia. An important character of the disease is the tendency of the symptoms to diminish in severity, and to disappear entirely after a sufficiently long removal from the cause which produced them.

Since, twenty years ago, chemical replaced sulphuric and chlorinated matches, physicians have had too many opportunities of studying in the workmen who make these articles, affections caused by phosphorus, affections consisting in necrosis and caries of the maxillary bones, and having this peculiar feature, that they are invariably localised in these bones, and never show themselves in any other part of the skeleton. The lesions of the bones then, which result from poisoning with phosphorus, have characters altogether peculiar and specific.

Gentlemen, in the specific diseases produced by physical or chemical agents to which I have now directed your attention, we can lay hold of and see the morbific cause: we can also lay hold of it, as it were, in virulent and poison-diseases. We know that morbid poisons exist in fluids secreted by persons labouring under diseases: the virus of rabies exists in the saliva of the mad dog, and the virus of small-pox in the pustule, although the fluids containing these poisons are identically similar in appearance to the fluids which produce no specific effects. We know that a morbific cause exists in particular secretions of certain plants and animals; for example, in the venom secreted by the gland placed at the base of the hooked dart of the rattlesnake, and in the juice secreted by the glands at the base of the hairs of the stinging nettle. Though in the majority of diseases we cannot thus, as it were lay our hands on the morbific cause, we are nevertheless entitled, as in natural history, to admit its existence. If we found a plant for the first time in a certain district, a plant till then unknown in that district, and if we afterwards found in the same locality a great number of plants all presenting precisely the same characters, we should be entitled to affirm that they all proceeded from one identical seed, although we had not actually seen that primitive seed. No comparison could in my opinion be better chosen, for nosological have, very properly, been likened to vegetable species: the living body has been considered as a field, in which, under certain conditions

inherent in the body, morbific seeds germinate, which spring up with their specific characters, reproducing the species, like the seeds of different species of plants confided to congenial soil. This comparison is perhaps more applicable to inoculable contagious diseases than to others, for of them we may with strict propriety say that the seed is sown, and the original reproduced; but it also applies to infectious diseases. When we see infectious diseases always characterised by similar symptoms, we are led to recognise the existence of special causes to account for the special effects, although we cannot actually lay hold of these causes; just as, in the illustration I have already employed, we are constrained to admit that all the plants came from the same seed.

Thus, Gentlemen, we all believe in the existence of what are called miasmata, although we can only form an opinion of them by their effects: we admit that there are several species of miasmata, because there are corresponding phenomena, peculiar special and invariable, which characterise the diseases which we suppose originate in them. Could any of you mistake marsh fever, which is generally characterised by intermittent paroxysms varying in type, and sometimes by neuralgic affections? Could any of you seeing such a case fail to conclude that the patient had been exposed to marsh emanations?

But here, although the morbific cause elude observation, we are at least acquainted with the conditions under which it has been developed. It often happens, however, that these conditions are not known; and yet circumstances convince us that a special cause exists in which originate the special effects which we observe.

We are not acquainted with the meteorological and terrestrial conditions under which cholera occurs, and our ignorance as to the cause of that disease, is still greater: nevertheless, no one seeing the disease invariably manifesting the same phenomena, will deny that it has a specific character. We are not acquainted with the cause of dothinenteria, but yet its specific character is admitted by every physician who sees it constantly presenting the same symptoms during life, and the same special anatomical lesions after death: these specific characters are so precise and predominant that confusion is impossible. Every one can distinguish dothinenteric from simple enteritis, when he has before him the anatomical lesions; and during life also, the symptoms enable the one to be diagnosed from the other.

To sum up, Gentlemen, the remarks which I have now made:—

we must consider that in every disease, there is a common element which may be termed the physiological element—inflammation, irritation, &c.; and likewise that which may be termed the nosological element, imprinting itself upon the former, giving to the whole disease a special character, and assigning to it a unity of origin, a special principle, a nature more or less determined, and, in a word, constituting a morbid species.

The common element predominates in diseases which are accidental: a burn produced by fire is an absolute type of this class. Here, quantity of morbific cause is everything, allowance being made for difference of organs and diversity of organization. Although in a great number of diseases, the nosological element dominates over the common element, it would be as absurd to exclude quantity of morbific cause from all participation in the production of effects, as it would be not to take into account diversity of organs and variety of organization: but quantity of cause, diversity of organs, and variety of organization are here all dominated by quality of cause, and, therefore, we require chiefly to consider the nature of that quality.

In certain cases, we can lay hold of the special cause, and, almost at will, produce the effects which are its natural consequences. So it is, in respect of the special phlegmasiæ produced by special physical and chemical agents, the diseases originating in a virus, in the poison of venomous creatures, or in the absorption of any poison: so it is also, in respect of certain diseases with the causes of which we are not acquainted, but with the conditions under which the causes act we are familiar, as for example, in marsh fevers. In these cases, the existence of a specific cause cannot be disputed: and it is not less present in other diseases in which the causes, as well as the conditions under which the causes act, elude our observation: the specific cause is established quite as well by the invariability of the symptoms and forms of the affection, as if we were also equally cognisant of the effects and the causes: from the constancy of the one, it is logical to infer the constancy of the other.

Gentlemen, perhaps some of you may think that I have already spoken at too great length upon the subject of the specific element in disease; and may be of opinion that its discussion would be more appropriate in a course on general pathology than in my clinical lectures. I have not, however, been at all afraid of going out of my province in thus discoursing to you, for though it be perfectly

true that the subject belongs to the domain of pathology, it is equally certain, as I have already remarked, that it meets us every moment at the patient's bedside, inasmuch as it is dominant throughout the whole of practical medicine. Its clinical importance appears to me to be so great, that I desire still to make some additional remarks upon it, with a view to show you how useful, nay how necessary for diagnosis, prognosis, and treatment it is, to understand the specific element of diseases. By placing before you additional details, I shall show you that a knowledge of the specific element in disease is the key of medicine, without which it is impossible to proceed successfully in the practice of our art.

To deny the existence of nosological species, or, in other words, not to take into account the quality of the morbific cause, and to consider only its quantity, to subordinate the nosological to the physiological element, is to maintain the uselessness of every differential diagnostic except that which is limited to the determination of the state of the organ affected, and the extent of the affection; since the nature of the disease, varying only in degree without changing its species, is necessarily known.

To push the argument to its ultimate consequences:—what advantage is there in seeking to distinguish small-pox from measles, if the pustular eruption which characterises the former is only a degree of inflammation of the skin more advanced than the exanthem which characterises the latter? The partisans of the dichotomic schools—if any such persons exist in the present day—would refuse to push the doctrine as far as this. When such persons have to do with diseases manifesting themselves by cutaneous eruptions, their first anxiety is to discover whether they have a case of small-pox, roseola, measles, or scarlatina: in spite of themselves, they admit the specific element, for their diagnosis is based on the specific characters of the eruptions.

If all accept this principle in respect of diseases, the anatomical manifestations of which appear on the skin, I ask, why was it necessary for Bretonneau and his pupils, physicians and surgeons, to use such great exertions to generalise and apply to other diseases, the doctrine of specific causes? Why, I ask, in the different phlegmasiæ, in those for example affecting the mucous membranes, has there been so obstinate a determination to see inflammations identical in their nature, and varying only in seat and degree?

Thus, according to the system which I am now combating, dothin-

enteria and dysentery are both forms of enteritis, as are also intestinal catarrh, colitis, and other inflammatory affections of the intestines produced by the action of sulphuric acid, arsenic, croton oil, or any other irritant poison. The anatomical characters of these diseases is essentially different; and do what you will, you will never be able to produce by means of sulphuric acid the lesions produced by arsenious acid, or croton oil; and still more obvious is it, that by none of these agents could you produce the lesions of dothinenteria. In respect of the other characters, the existence of the specific element is still more conspicuous. Dysentery and colitis possess a similitude in kind: both are ulcerative inflammations of the large intestine; but still they have characters so distinctive that it is impossible to mistake the one for the other. I shall have occasion to point out their respective characters during the course of these lectures.

The same remarks are applicable to affections of the respiratory organs. In the simplest catarrh, in whooping-cough, and in asthma, the dichotomists see only bronchial phlegmasia, and do not stop to consider the individual peculiarities by which they are distinguished from one another. When I come to speak to you of these different diseases, I shall take care to describe their characters; but for the present, let it suffice to say, that it is of the utmost importance to be acquainted with them, so as to be able to avoid confounding simple enteritis with follicular enteritis or dothinenteria; or whooping-cough and asthma with simple bronchitis.

The importance of this kind of knowledge is very great in relation both to prognosis and treatment. I have already called your attention to this point when speaking of dothinenteric catarrh of the intestinal canal. I then told you that that affection was one of great danger; that the progress of simple enteritis and dothinenteric enteritis was quite different; and that when the practitioner is not acquainted with the natural course of each species, he cannot form a correct prognosis. Take another example. A patient comes with sore throat: he states that on the previous evening, consequent on a chill, he was seized with general discomfort, pains in the back and limbs, rigors, loss of appetite, and fever. Next day, he complains of difficulty of deglutition; and there is swelling, but only slight swelling, of the submaxillary glands. Upon examination of the pharynx, it is found that there is swelling of the tonsils, with redness of the pillars of the veil of the palate; and that a secretion

having exactly the appearance of false membrane covers the affected surface. Let us suppose that you were sent for at the same time to see another patient also affected with plastic sore throat, but in whom there was a different development of the disease. Without any appreciable cause, he had experienced for some days general uneasiness accompanied by fever: the sore throat was much less painful in this patient than in the other. If you only take into account the anatomical element common to both cases, the resemblance between the two is complete. The scalpel, the microscope, and chemical analysis will all demonstrate, that in both cases the false membranes are identically the same: to judge from appearances, the last-mentioned patient seems the least unwell of the two. But if you allow the diseases of both to run their course without interference, you will see that the malady which set in with the greatest violence and most acute pain, and was accompanied by a degree of fever absent in the second case—you will see, I say, the acute sore throat get quite well spontaneously and quickly, leaving no trace behind; while the other may carry off the patient with symptoms of general poisoning, or by suffocation consequent upon the development of pseudo-membranous laryngitis or croup. In both cases, however, there was plastic sore throat; but with this difference, that in the one case the malady was common membranous sore throat, that is to say herpes of the pharynx, which is seldom a serious disease, while the other was malignant membranous sore throat—diphtheritic sore throat—which is, as a general rule, a very formidable malady. You see then, Gentlemen, that under such circumstances, as I have now been supposing, it was important to be acquainted with the specific character of the two diseases so similar in appearance; for you might in the one case mistake a malady naturally benignant, for one of formidable character, and in the other you might prognosticate a mild attack, while the case was destined to terminate in death, or in a long and chequered convalescence, retarded perhaps by paralysis more or less general, and more or less persistent.

I need not on this occasion multiply examples to illustrate this point; for we shall only have too many opportunities of returning to the subject of the specific element of disease, which, as I have said, constantly presents itself in the course of the clinic. I now come to that aspect of the subject which bears on therapeutics.

Gentlemen, to cure, and, when that cannot be done, to alleviate

the sufferings of patients is the object of medicine. The fact that the term *medicine* is derived from the Latin verb *mederi*, signifying *to take care of, to apply a remedy, to cure*, sufficiently points out the nature of our mission. Therapeutics, as it comprises the study of the means by which we hope to carry this out, is consequently the most important department of our art: and, as you know, it is by far the most difficult. While treatment is dependent upon the experience, talent, and tact of the physician, it is still more subordinate to the nature of the disease which he wishes to cure, to particular conditions under which the disease exists, to the peculiarities in the organization of the patient, and to a host of circumstances too frequently unknown. The treatment of diseases necessarily rests upon a knowledge of their symptoms, but it is also based in a special manner upon a knowledge of their causes and their natural history; and it is from the latter description of knowledge that a just appreciation is obtained of the important part performed in disease by the specific element.

How is it possible to estimate the value of a method of treatment, or form an opinion as to the efficiency of a remedy, if the operations of nature—to use an expression of our predecessors—are ignored, operations which are different in the different species of diseases? By not discriminating between these different species, do we not incur the risk of attributing great virtues to medicines which have in reality no remedial power, and of refusing to admit that others possess any therapeutic properties, although their utility, when administered under suitable circumstances, is undoubted.

It is thus that we explain the fact that some have extolled pretended substitutes for cinchona, while others have blamed cinchona for transforming intermittent fever into malignant dothinenteria. The former had to do with simple cases of common continued fever, which would have got well of themselves, and which were at the commencement invested with the intermittent form; the latter had to do, not with marsh fevers, but with cases of dothinenteria which had an intermittent type at the onset—such were the cases of fever the fatal progress of which was not arrested by cinchona. This is a topic to which I have already directed your attention when lecturing on dothinenteria.

In the same way, if a simple colitis accompanied by bloody stools is mistaken for dysentery—a mistake which I see committed every day—it is impossible to avoid very erroneous conclusions in thera-

peutics. It is supposed, for example, that dysentery has been cured by a few leeches and emollient enemata, because in the cases in question there existed profuse bloody discharge, frequent stools, much straining, and high fever; whereas, in reality, the affection was one from which there would have been recovery in a few days, without any treatment whatever having been employed. Confronted with a case of real dysentery, the practitioner applies the same treatment he followed, as he thought with such amazing success in the case of colitis which would have got well had he refrained from all treatment; and he is astonished at its failure.

You are called in to a patient suffering from great dyspnœa. His respiration is accompanied by a whistling in the larynx, which at once attracts your attention: on carrying your finger behind the base of the tongue, you discover that there is swelling of the epiglottis and aryteno-epiglottidean ligaments; on making pressure over the larynx, pain is caused. You are told that the person began to lose his voice two or three months ago, and that from that period, it had become feebler and feebler, till at last there was complete aphonia. Inspiration, at first whistling only during sleep, or after walking rather quickly or ascending a stair, has become similarly characterised when the patient is in repose: the oppression of the breathing has increased so rapidly, that when you are summoned, you see that unless a change for the better very soon take place, tracheotomy will be the sole remaining means of preventing death. Upon inquiry, you learn that the œdema of the glottis, depending upon serious lesions of the larynx, the cartilages of which are perhaps necrosed, or at least the mucous membrane of which is ulcerated—you learn that the laryngeal affection was some considerable time preceded by other local symptoms. The individual is stated to have had chronic coryza, characterised by a bad nasal discharge, to have thrown off crusts from the mucous membrane of the nose, from which organ a fœtid odour is exhaled: you find that he has also had tumours on the bones. Without proceeding any further, you diagnose syphilis, and forthwith institute a system of treatment under which recovery proceeds. From the suffocative seizures having been of so formidable a kind as to place the life of the patient in imminent peril, you have performed tracheotomy; and in doing so, you have been aware that your operation, by retarding death, justified the hope of the patient being restored to perfect health.

By a fortuitous concurrence of circumstances, such as often happens

in practice, you may at the same time have been sent for to attend another patient also affected with œdema of the glottis, but in whom you have found the disease associated with the tubercular diathesis. Now, if in the latter case, taking into account only the condition of the larynx, if, ignoring the specific cause of the disease, you try to obtain, by the same means, results similar to those obtained in the other case, you will inevitably fail.

You may see in the same ward of an hospital, three patients with neuralgia of the fifth pair: in one, the paroxysms return every day, and are characterised by horrible pains which continue for six or ten hours accompanied by lachrymation, coryza, and salivation: in another, the neuralgia returns four or five times in the twenty-four hours, is accompanied by the same phenomena as in the first case, but only lasts for two hours: in the third patient, the fits recur at least every two or three hours, and last at the most for a minute, but they occasion agonising pain, and are accompanied by convulsive movements of the face. The three affections are apparently similar, and occupy the same seat: the first, being an intermittent fever in a metamorphosed form will yield to cinchona: the second will be advantageously attacked by preparations of iron, if connected with a chlorotic condition of the patient, or by veratria, colchicum, or the external use of belladonna, if the case is rheumatic neuralgia consequent upon a chill: but the third will resist every kind of treatment, for it is tic douloureux or epileptiform neuralgia.

A great many similar facts might be adduced; but from those now stated, you can understand, that in treating diseases, it is absolutely necessary to bear in mind their specific element. I must state, however, that in some cases a knowledge of this element proves of very little consequence. In eruptive fevers, for example, following their regular course, the intervention of art is either quite useless, or very rarely of any benefit.

I have hitherto spoken only of the specific character of diseases; and now I have a few words to say regarding the *specific properties of medicines*. We need not be long detained by this subject, if we adopt Parr's definition of specific remedies, and understand by that term only those medicines, which, like quinine in intermittent fever and mercury in syphilis, produce always, and in all patients, the salutary effects attributed to them—acting upon the malady in virtue of an unknown power, attacking in a direct manner its very essence, without its being necessary for the prescriber to take into

account the form in which the symptoms appear. We should very soon exhaust the list of specifics, were we to restrict in this manner the application of the term; for there is not a specific remedy for every disease which has a specific character. Again, in practice, we do not always find specific remedies so efficacious as we have expected them to prove. Indeed, it sometimes happens that medicines very justly called specifics not only fail, but even aggravate a malady, which, judging from their usual action, they ought to have cured. In such cases, we must abandon them, and have recourse to the use of remedies called *rational*, or in other words, to those which are indicated by the symptoms.

Two women, who, at an interval of some months, successively occupied the same bed in St. Bernard's ward, furnished facts in support of this proposition. They both had syphilis: mercury administered according to rule, and in a very guarded manner, had checked the progress of the symptoms, when it became necessary to suspend the use of the medicine: the patients had fallen into a very bad state of chlorotic cachexia, rendering obligatory recourse to preparations of iron, under the influence of which their health was rapidly re-established. In other patients you will see still more formidable symptoms arise: you will see an extension of ulcerations which the mercurial treatment ought to have cicatrised; at other times, the alimentary canal becomes irritable, fever is set up, and a pseudo-syphilis supervenes, complicating and altering, without curing, the true syphilis.

In short, Gentlemen, the action of specific remedies does not materially differ from the action of those called rational. The curative action of both is preceded by a vital action which they excite: this may be called their immediate or physiological effect. The difference between their operation consists in the specifics exerting a special and direct influence upon the pathological actions which they modify, their immediate effects merging into the remote or curative effects; while in respect of the remedies called rational, the two kinds of effects appear distinct from one another.

Without stopping longer to consider this scholastic distinction, suffice it to say, that medicines which modify the organism in a pathological state, in the same way that hygienic agents modify the organism in a state of health, have properties common to the whole class of medicines to which they belong; and only excite in the economy a common or general action, such as stimulating or depress-

ing, irritating or calming. But along with these common properties, they each possess special properties which produce specific effects; and the two kinds of properties, inasmuch as they exist in very variable proportions, also manifest themselves in very different ways, according to the individual predispositions of the subjects to whom the medicines are administered. This is what I understand by the specific action of medicines.

To develop fully this subject, which embraces the whole domain of therapeutics, would carry me far beyond the limits which I have prescribed for myself, as it would oblige me to review, if not all medicinal substances, at least all systems of medicinal treatment. I refer you, therefore, to the treatise on therapeutics which I have published in conjunction with my colleague and learned friend Dr. Pidoux; and in particular I ask you to read that portion of it in which we speak of substitutive treatment [*médication substitutive*], a method of proceeding entirely based on the existence of a specific element in disease, the doctrine which we have now been briefly considering.

LECTURE XXIII.

CONTAGION.

Definition.—Parasitical Diseases are not included.—Spontaneous Development of Morbific Germs.—Infection.—Infectious Diseases may become Contagious.—Dormant State of Germs.—Difference between Infection and Contagion.—Morbific Matter.—Conditions of Contagion: inherent in Individuals and in Germs.—Immunity, Temporary and Absolute.—Conditions as to Age, and Previous Contamination.—Acclimation and Habit.—Apparent Immunity.—Modes of Transmission.—Contact.—Direct Inoculation.—Inhalation.

Gentlemen:—The question of contagion is so intimately connected with that of the specific element in disease, as to form its necessary complement.

The term *contagion* has been very variously defined; but the definition which seems to me to be the most accurate is that of Dr. Anglada of Monpellier. It may be objected to on account of its length, but if this be a fault, it is one which must be attributed to the nature of the subject, and not to the author: in fact, it is on account of its completeness, that I prefer it to other definitions.

Contagion, adopting the definition of Dr. Anglada, I regard as:—"the transmission of a disease from one person affected with that disease to one or more other persons through the medium of a material cause [*principe matériel*], the product of a specific morbid elaboration: this material cause communicated to an individual in a state of health determines the same phenomena and symptoms in him as were observed in the individual from whom the germ proceeded."[1]

[1] Anglada:—Traité de la Contagion, pour servir à l'histoire des Maladies Contagieuses et des Epidémies. T. i, p. 12. Paris: 1853.

The necessity of the transmitted material cause being elaborated excludes from this definition parasitic diseases, which have been considered contagious by some physicians. In fact, itch, porrigo decalvans, thrush, &c., though communicated from person to person, cannot be looked on as contagious affections. Were we to admit that the acarus scabiei, trichophyton tonsurans, and oidium albicans are transmissible by contagion, it would likewise be necessary to hold that the parasitic animals which infest the exterior of the body, such as bugs, fleas, and the different kinds of lice, are also similarly communicated. But it has never occurred to any one to maintain such a proposition. I grant, however, that there is a certain analogy between parasitic and contagious diseases; for while it is impossible for any one to say that lice are contagious, such a statement might be made with some verisimilitude in respect of the trichophyton of porrigo decalvans and the oidium albicans of thrush. Pushing matters to the extreme, it might then be alleged that the contagium of small-pox is simply a parasite, which, like the oidium albicans is transmitted from one person to another. I anticipate the objection which will be taken to this line of argument, and I confess that I am in a rather awkward position to reply to it, as I hold that contagious diseases sow themselves by seed, and are consequently transmitted by germs. Still, I stand out for the distinction which I have drawn, maintaining that it is established by the capital fact that there is this difference between contagious and parasitic diseases, that in the former, the material morbid cause eludes my observation, while in the latter, I can lay hold of it. I can see, and I can isolate the mycelium of thrush, the tricophyton of porrigo decalvans, and the acarus of scabies; and, placing them in the field of my microscope, I can study and describe their characters. This I could not do with the morbific germs of small-pox, measles, or scarlatina, which, unlike the parasites, have not an independent existence, but require an organised and living substratum, to enable them to exist, and to show that they exist.

Other affections, which by an overstrained employment of the term, have been called contagious, are also excluded from Anglada's definition. Every day I hear people say that laughing and yawning are contagious. The expression must be regarded as only a figure of speech: according to the same phraseology, certain nervous diseases are contagious. Who does not know the history of the women of Abdère, of the nuns of Loudun, of the choreomaniacs of

the middle ages, of the convulsionaries of St. Médard, and a hundred other similar histories, which have been a hundred times told. In these cases we cannot, speaking the language of medical science, use the term *contagion*: we must employ the word *imitation*.

Infection, when used to signify a morbific cause, is frequently employed to point to something different from, or in contrast with contagion: it differs from, but does not exclude contagion. Frascator was the first author who thoroughly appreciated this distinction: his researches into the nature of syphilis led him to study the question. In his work "*De Contagionibus*," he wrote these words: "Qui hausto veneno pereunt, infecti esse dicimus, minime autem accepisse contagionem."—Of those who die after taking a poisoned draught, we say that they are infected: we do not say that they have received contagion. Frascator established the differences which he pointed out: I am also now going to establish them.

A person is stung by a wasp, or bitten by a serpent: the venom introduced into the system forthwith produces symptoms which, according to the circumstances, are more or less serious, or fatal: this is infection. If it be objected, that the germ of the disease coming from an animal and being by it transmitted to a man, contagion might be averred according to Dr. Anglada's definition, I reply by quoting the very terms of the definition itself, from which it appears, that while there has been transmission of a germ developed within a living organism, that transmission has not taken place from a sick to a healthy individual, nor has there been anything morbid in its elaboration within the animal which produced it. To use Frascator's expression, there has been a poisoned draught, and nothing more: the manner in which the *haustus*, or the absorption has taken place is of no consequence. A person suffers from symptoms resulting from unwholesome diet, from the daily use, for example, of flour containing a certain admixture of ergot of rye: in such a case, could it be said that there had been contagion? No: but it could be correctly stated, that there had been infection. Or again, in place of entering the system through the medium of the digestive organs, the infection may have effected its entrance by the respiratory passages, as takes place in diseases occasioned by various deleterious gases. Here again is the poisoned draught: be it venom, poison, or deleterious gas, you can take hold of the morbific cause; but there are other cases in which the cause is quite unknown.

Take the case of a man living in the vicinity of a marsh, and let us suppose that the ground in the neighbourhood of his dwelling has been recently turned up: the most acute sense of smell cannot detect any unpleasant odour, vegetation is everywhere luxuriant, the air seems in all respects salubrious: the man nevertheless is attacked by illness—by intermittent fever. He has been infected by a morbific germ contained in that air apparently so pure: the germ, though it only reveal itself by its effects on a living organism, does not the less certainly exist. In this case, we should not say that there had been contagion, but that there had been infection; for here, as in the other illustrations which I have adduced, there was no transmission of a disease from a diseased to a healthy person: the morbid cause which engendered the malady was not the result of a morbific elaboration within another animal. The definition of Dr. Anglada is sufficiently comprehensive to meet all these differences.

When the diseases which are designated "infectious" originate under the conditions which I have just been pointing out, under conditions of a manifestly vitiated atmosphere, as well as when the vitiation is not cognizable by our senses, we say that there has been infection. But our science is completely at fault, when, without any apparent change in the telluric or atmospheric conditions of a locality, there supervene what are called epidemics.

Thus, at the end of March, 1832, when cholera came for the first time to commit its ravages among us, the weather was cold and dry, with beautiful sunshine: there was no apparent change in the geological constitution of the soil, nor in the meteorological constitution of Paris; and yet the disease as soon as it was developed spread with frightful rapidity. If transmission by contagion had been given as the explanation of this rapid spread of the disease, it would most certainly have been at once confuted by the manner in which the epidemic dealt its first blows. It would have been necessary to seek the explanation in some general influence existing in the external world; or in other words, to admit infection, without being able to demonstrate the cause.

There is another circumstance which it is necessary to take into account. If a malady believed to be contagious is only propagated in the same place, and does not extend beyond the locality, even when a large number of contaminated persons are assembled together and are in contact with healthy persons, the contagiousness of the disease is disproved, and we say that it is only infectious. But I

have often asked myself, whether sojourn in an infected place does not induce a predisposition in virtue of which the slightest contagion might act energetically, although the same contagion increased tenfold in power would be incapable of affecting an organism not predisposed by local infection. I have always been astonished at the immunity sometimes enjoyed in a town where there prevailed an epidemic reputed not to be contagious, by persons who carefully avoided communication with the sick.

Here, Gentlemen, arises the great question of *spontaneous origin of epidemic and contagious diseases* simultaneously affecting large numbers of persons.

Can diseases really arise spontaneously? Or, are they in some sort of way innate in the human species? Are they, as our predecessors said, originally present, their power remaining *in posse*, ready to manifest itself, waiting to enter *in actu*, upon favouring circumstances arising? Some physicians adopt this latter proposition: in their opinion, the germs of disease are coeval with the human race, every individual having them in his body, *apothecam hoc virus recondentem quivis homo in se gerit*, and they suppose that sooner or later these germs develop *fermentem morbosum, nunc citius nunc serius actuosum redditur*. This opinion, in former times maintained by men of the greatest eminence, though opposed by others of equal repute, has still some supporters. It does not, however, require a prolonged study of the question to side with those who deny the pre-existence of morbid germs, and believe in their spontaneous development. To arrive at this conclusion, it is only necessary to remember that some of the most contagious diseases, such as pox and small-pox, were unknown to Hippocrates, Celsus, Aretæus, and Galen, and consequently, were non-existant in the times of these great observers. As I formerly remarked, when discussing this subject in relation to vaccination, they could not possibly have failed to describe diseases possessing such precise characters, had they seen them. Pox, as you are aware, was not very well known, till after the fifteenth century, although historians mention its existence in the times of the crusades. There is no positive mention of small-pox till the 7th century, as is stated by Sprengel in noticing an epidemic of that disease which occurred in 565, and another which occurred in Arabia in 572.[1] Is it possible

[1] Sprengel:—Histoire de la Médecine depuis son origine jusqu'au 19e siècle; traduite de l'allemand par Jourdan. T. ii, p. 198, 199: Paris, 1815.

to believe that the germs of the disease were coexistent with the human race, and remained for so many ages in a state of incubation?

Spontaneous development, then, in respect, even of the most contagious diseases, must be admitted. As contagion necessarily implies the presence of two individuals, the one the giver and the other receiver of the morbid germ, it is a truth, so self-evident as not to require to be stated, that in the first sufferer from the disease, its origin must have been spontaneous, though wholly under the influence of unknown causes.

While there is reason to believe that at present some diseases, such as syphilis, small-pox and measles, are always reproduced by contagion, that that is now their sole mode of originating, there are other maladies which we constantly see arise spontaneously. Does not rabies become developed in animals of the canine and feline species under the influence of particular causes, irrespective of any contagion or antecedent inoculation? The cases are numerous and indisputable. It is so likewise with the malignant carbuncle [*sang de rate*] in animals of the ovine species: this disease is spontaneously developed by sheep under the influence of certain telluric, atmospheric, and alimentary conditions. Interesting observations made by physicians of the department of Eure-et-Loir, an account of some of which you will find reproduced in the painstaking work on anthrax carbo by Dr. Raimbert of Châteaudun, establish this fact in a most conclusive manner;[1] and also show that special conditions of soil and air were inherent in the localities where the *sang de rate* was decimating the folds, while the disease did not show itself in other districts unless imported into them by infected sheep.

But whatever may have been the causes which originated these diseases, they have the power of reproducing themselves by contagion. The morbific germ, which in its first generation was of necessity spontaneous, reproduces itself within the body, and, in its turn, furnishes other identically similar germs capable of continuing the morbid species, always producing in the individuals who receive them effects the same as those which were manifested in the individuals whence the germs came; and being capable in the same manner, without any change of character, of perpetuating themselves in indefinite succession.

[1] Raimbert:—Nouveau Dict. de Médecine et de Chirurgie Pratiques. Article, Charbon. T. vii, Paris: 1867.

For the accomplishment, however, of this transmission, it is necessary that it should take place between individuals of the same species. When there is diversity of species, the germ either ceases to be transmissible, or, when it does pass from the one species to the other, it produces different effects.

In relation to the first point:—rabies, for example, is communicable from the dog or cat to man, and from the dog to other animals, producing in all of them symptoms similar to those observed in the animal from which they were transmitted; but there stops its capability of transmission: it is only communicable by man and individuals of the genera *canis* or *felis*. In 1826, during my *internat* at Charenton, I several times received on the face, lips, and eyes, the saliva of patients affected with rabies, without any resulting inconvenience. Recently, also, my *chef de clinique*, Dr. Dumontpallier, having punctured himself with an instrument which he had used in making the autopsy of a patient who had died of rabies, did not experience any consequences from this accident, though he dreaded their occurrence. M. Raynal of Alfort inoculated several dogs with the saliva of the same patient collected both before and after death; but in none of the cases was there any result.

In relation to the second point;—let me recall to your recollection the remarks I made, when lecturing to you on vaccina, regarding the transformation of the disease in horses called grease [*eaux-aux-jambes*] into cow-pox, and of cow-pox into vaccina; and let me also recall to your recollection the mutation of ovine malignant carbuncle into anthrax carbo and malignant pustule. It would appear that in these cases, by being cast into a particular soil, the morbific seed is changed, and the resulting species modified: so obviously is the change dependent on the nature of the soil or substratum, that malignant anthrax inoculated from cow to sheep—inoculated under certain conditions—appears in the latter as ovine malignant carbuncle [*sang de rate*].

Let us now resume the subject of infection. In whatever manner infection takes place, whether it be by a miasm or a virus, or by the agency of an unknown cause, contagion is not excluded. I have just proved to you that rabies and ovine malignant carbuncle undoubtedly become contagious, and I could establish the same proposition in respect of other diseases which like them are from the first infectious.

Dysentery and the typhus of camps are striking and unchalleng-

able examples of this. Our latest and glorious campaign—the Crimean campaign—unfortunately furnished us with a new opportunity of judging the merits of this question. The typhus which so cruelly struck down our soldiers was, as is usual, developed under the influence of overcrowding, or to speak more correctly, under the influence of the assemblage of a large number of men in one place. The morbid germ, produced spontaneously amid conditions belonging to the external world, and elaborated within living organisms passed by contagion to, and produced typhus in, other persons who had not been subjected to the same conditions as those originally attacked: through the sole influence of contagion, the typhus seized the victims, not only in the country where it arose, but likewise in countries distant eight hundred leagues: it was brought among us by invalided soldiers, and attacked persons who had never left Paris. As you know, Gentlemen, the nursing sisters and servants at the military hospital of Val-de-Grâce fell under this scourge when ministering to soldiers of our army of the East who were under treatment for typhus in that institution. You will find the facts to which I refer stated in a work on the subject by Dr. Godelier, professor of Clinical Medicine at the Val-de-Grâce.[1] Thus you see that typhus which was originally caused by infection, ultimately becomes quite as contagious as small-pox. The same statement is true in respect of dysentery and other epidemic diseases.

It is necessary, however, to guard against a misunderstanding of this question. Sometimes, diseases are regarded as infectious which are exclusively contagious. This mistake arises from not investigating into their starting point, or from not being able to discover it. In this way, the view that they are not contagious, and have been spontaneously developed, is adopted. No doubt, as I have remarked, these diseases were at some particular period produced under influences totally unconnected with contagion; but that period is very remote from the present time, and since that time, whenever it may have been, they have always, as now, been reproduced by contagion.

I admit that it is often exceedingly difficult to discover the source of the malady. A person takes small-pox: in spite of all the care with which you set yourself to find out where he contracted it, you fail to do so: the patient tells you positively that he has seen no

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[1] Godelier:—Mémoire sur le Typhus observé au Val-de-Grâce de janvier à mai, 1856. [*Bulletin de l'Académie de Médecine, T. xxi, p.* 887.]

one who had the disease, that in the house where he lives, among his acquaintances and among all with whom he comes in contact, he knows of no case of small-pox. You then say the disease has been spontaneously developed. But this individual has perhaps touched the garments of a man who died of small-pox; perhaps he has gone into a room where there had been, at a more or less distant date, persons suffering from small-pox. The contagion of the malady, difficult to demonstrate in the great centres of population, can be more easily followed up in small places; on a former occasion, I was at some pains to establish this point.

Even in Paris, however, we sometimes have an opportunity of tracing back the contagion to its source. In 1827, I attended in the rue de l'Echiquier, in Paris, a young woman with small-pox. She lived with her mother, a poor linen-draper. Both women inhabited the ground-floor, which consisted of one room divided by a high screen. The division next the street was the shop, in the compartment behind the screen was the one bed in which mother and daughter slept. During the entire duration of the case of small-pox, the neighbours came as of wont to make their purchases, and none of them had any suspicion of the danger which they thus incurred. At that time I lived in the rue de l'Echiquier; and so was enabled to watch carefully the development of a perfectly local little epidemic. In less than six weeks, seventeen of the patient's neighbours were attacked with small-pox: and I ascertained from the mother, that the persons first seized in each family were persons who had come to make purchases at her shop. Now, as no one knew how the disease had spread, the different medical men who were called in remained convinced that it had been spontaneously developed.

It is necessary to enter still further into details, so as to enable me satisfactorily to explain my opinions.

In 1854, *the Wellington*, an English ship, sailed for the east, having on board a regiment of infantry. Some days after leaving port, small-pox broke out, and in a short time a great many soldiers were infected. This ship put back to Plymouth, where she was thoroughly cleaned in every part, and in fact made as good as new. Some time afterwards, when supposed to be quite purified, she sailed for the Crimea with troops. After being fifteen days at sea, small-pox reappeared on board, and made new victims: cases also occurred among the wounded whom the Wellington brought back to England

from the Black Sea. A second time, this ship was subjected to purifying processes: it was supposed that every possible precaution had been taken, and it was hoped that she had been rendered a salubrious habitation: nevertheless, on her third voyage, the disease declared itself a third time. It matters little how the first epidemic was developed: but let us examine into the source of the disease in the second and third voyages. When the first soldier took small-pox, more than nine days, that is to say more than the ordinary period of that disease's incubation had elapsed since the ship had left England, so that one might come to the conclusion that the pestilence had been spontaneously developed. But, would it not be more reasonable to infer that the Wellington had retained contagious germs since her previous voyage?

Has not Dr. Mélier, in his learned report on the yellow fever which raged at St. Nazaire in 1861, proved that the ship Sainte-Marie, from the Havannah, was the source of the contagion of the yellow fever which broke out among the men employed in unlading her at St. Nazaire?[1] He has shown you the disease, transported to a great distance from its original home to a new locality, and there passing by contagion from man to man. My honourable colleague, Dr. Chaillon, fell a victim to it, having contracted the contagion from remaining some hours in attendance upon one of the workmen engaged in discharging the cargo of the Sainte-Marie.

During last century, there was ordered the judicial exhumation of a person who had died of small-pox a great many years previously. The grave-digger who performed the exhumation, and some persons who were present when it was being done, took the disease: it soon afterwards broke out in the little parish in which occurred the events now mentioned, and where for many years small-pox had not been seen. This history has an apocryphal appearance; but, nevertheless, it is related by authors fully deserving of credit. It teaches us that the variolous germ, wrapt in a shroud so to speak, but in reality only deposited on the planks of a coffin, was capable of affecting a considerable number of persons, and of developing itself with formidable energy whenever it met with conditions favourable to development. The preservation of the morbid cause on board the Wellington is a not less credible fact.

[1] Mélier:—Relation de la Fièvre Jaune survenue à Saint-Nazaire en 1861. [*Mémoires de l'Académie Impériale de Médecine. T. xxvi. Paris*, 1863.]

Morbific germs may remain inactive for a certain time, adherent to inorganic bodies, as is illustrated by the practice of former days of inoculating by means of a thread impregnated with variolous matter. In this way, they may remain hidden for days, months, or years, waiting to manifest their presence, till they meet with conditions favourable to their evolution.

Have not the experiments of Spallanzani and of Réaumur disclosed facts quite as extraordinary, relative to the development of animal and vegetable germs? Did not the first named of these illustrious inquirers into the secrets of nature observe the development of infusoria in dust collected from the gutters of roofs exposed to the rays of an intensely ardent sun? One drop of water sufficed to bring about the resurrection. And have we not recently been present at spectacles equally marvellous? Are not you all acquainted with the history of the seeds found in the tombs of the Pharaohs, which germinated and fructified after a lapse of more than three thousand years, just as if they had been gathered on the previous day from their parent plants?

Those among you who take an interest in botanical studies have observed a well known appearance presented by the flora of the woods. By the cutting down of the wood, this flora is so greatly modified, that after the interval of a year it is impossible to recognise it. In the situation where you previously found plants of a particular species, others of a totally different kind have appeared, which have not been seen since the first time the wood was cut down twenty-five years previously. During twenty-five years, the germs have remained buried in the ground, waiting for the air and sun requisite for their developing themselves. It may be said that the seeds have been sown by the wind, or that they have been brought from afar by birds, just as we see crows and magpies carrying kernels and nuts which they have gathered, and accidentally dropping them here and there. But how are we to explain the number and variety of the plants which appear under the circumstances which I have described? In particular, how are we to explain the fact that according as the wood is thick or cut down, we always have the one and not the other kind of plants?

Let me now return to the consideration of morbid germs. I have referred to what occurred long ago at Gibraltar. In 1802, the English troops, on their return from Egypt brought ophthalmia with them to Spain, a disease which had till then been unknown on the

coasts of the Peninsula. From that time, ophthalmia attacked in succession the different regiments which constituted the garrison of Gibraltar. Such at least was the state of matters in 1828, when I was sent there on a mission: the English surgeons showed me soldiers affected with Egyptian ophthalmia, although during the preceding twenty-six years the bedding and furniture of the barracks had been frequently renewed: everything had been done that could be thought of to improve the sanitary condition of the barracks.

Here is another case in point. The history of contagion abounds in such cases. In 1845, a woman was admitted to my wards at the Necker Hospital with all the symptoms of glanders, of which she died. Where did she contract this disease? She worked at the establishment of a merchant of horse-hair, where her occupation was to twist hair which came from Buenos Ayres. Mark well this circumstance: it is a fact of chief importance that all the hair in that establishment came from Buenos Ayres. Well! the woman contracted glanders, and the only way in which we could account for this was the nature of her occupation: she had never had the care of horses, nor had she ever had any communication with persons so employed. If there are any cases in which we can suppose that an infectious germ was spontaneously developed, this is certainly one of them; and yet, extraordinary though it be, the cause of the contagion seemed to me quite evident—the contagium of glanders existed in the South-American horse-hair.

Nor does this case stand alone: it is well known that glanders unfortunately too often attacks workers in horse-hair, just as malignant pustule attacks workers in wool. To me, and others, the facts now stated are irresistibly convincing, and prevent disbelief in the possibility of a prolonged conservation of contagious germs.

In following out the details of the evolution of these germs, I must constantly rest upon analogies, a proceeding always necessary when direct facts are wanting. I propose to take my analogies from natural history and agriculture.

Some seeds will grow anywhere. Place them under certain conditions in respect of heat and moisture, and they will spring up in all places and at all seasons. But there are other seeds which do not behave in this manner.

Make, for example, in February, a seed-bed of cherry-trees, casting a thousand cherry-stones into a thoroughly prepared soil. During April, you will see some stems coming up; if the twentieth

part of your seed spring up, you ought to be satisfied. In the following April, more of your seed will germinate; and again, in the April of the succeeding year, an additional quantity of your seed will arise. If, in these successive evolutions at intervals of twelve months, occurring always at the same time of year, you look to the influence of the seasons for an explanation, I ask—by appealing to what influence can you explain, why seeds placed under precisely the same conditions of soil, air, sun, and water have not germinated simultaneously?

The germs of some animals offer similar examples. Réaumur, wishing to study the habits of the *bombyx pavonia major*, kept several chrysalis of this kind of butterfly in the sand-box of his writing-table. Some of them hatched, and others seemed as if they were dead, till he touched them with the point of his penknife, when he found that he excited slight movements. He kept them: and next year, at the same period, almost to the very day, he saw an additional number of butterflies come forth: also twelve months later, and again almost to the day, a third hatching took place.

Is there not something very curious in this repose of germs? Is it not singular that chrysalis of the same butterfly, of the same age, and placed under precisely similar conditions, should have been hatched at intervals of exactly one and two years? Why may not the seeds of disease comport themselves in this respect like the seeds of plants and the larvæ of insects? Conditions of air, sun, water, and place can no more explain the successive evolutions of the germs of contagion than they can explain the successive hatchings of the larvæ of Réaumur's bombyx. It would appear that the germs of many diseases like the germs of some animals and vegetables are only developed at determinate epochs. Yellow fever, for example, has never prevailed in Europe except from July to September, whatever may have been the meteorological constitution of the other months of the year. The disease has always appeared within that period, whether it has been a period of heat or cold, of drought or rain. It has in this respect a peculiarity similar to that of some birds which always moult at the same season of the year, in whatever climate they may be living. The parrots of the southern hemisphere change their plumage in March, the time when the temperature begins to be lower in their native regions: they, when brought to France, remembering their origin, if I may be allowed the expression, still moult in March, though in our latitude, at that

season, the warm weather is only beginning, and though the birds of our country do not moult till September.

But it may be said that no one has ever seen these morbid germs of which I have been speaking: no one has ever collected any of the poisons, the absorption of which gives rise to cholera, yellow fever, influenza, intermittent fever, and dothinenteria. That is quite true; and yet the persons who deny the existence of the germs really accept the essence of the proposition, and take exception only to a term, for they speak of miasmata and morbid causes, which they have not seen any more than they have seen germs.

The recent labours of Professor Charles Robin, of which I shall speak immediately, seem to put us on the track to discover these germs of disease.

The miasmata, or morbific causes, or germs—the term matters little—may remain latent, slumbering for a longer or shorter period, buried in inorganic substances: then, at a particular moment, under certain telluric and atmospheric conditions, with the nature of which we are unacquainted, but the influence of which no one denies, they develop themselves in persons predisposed to receive them.

I ask those who refuse to admit the pre-existence and slumber of germs, if they have found the intervening conditions which alone they put forward, and under the influence of which, yellow fever, after having been absent from a district for ten, twenty or thirty years, all at once begins to rage with an intensity equal to that manifested at its first appearance ten, twenty or thirty years before. During this long interval, have you detected any change in the atmospheric constitution of the locality? Have its meteorological conditions appeared to be modified? Do not deny that germs exist, because their existence is incapable of direct demonstration; for you cannot prove in any more satisfactory manner the intermediate influences which you unhesitatingly admit. Have I not sufficiently established my proposition by citing the successive epidemics of small-pox on board the Wellington, and the installation of Egyptian ophthalmia in the barracks of Gibraltar?

If we admit the existence of germs, to argue against their slumber, to explain their spontaneous appearance, by alleging that they have been borne by winds from one country to another, would be to substitute for one hypothesis, another hypothesis based on entirely false premises.

Let me illustrate this point by referring to the yellow fever which

prevailed at Gibraltar. The winds, it has been said, carried thither the germs of the disease: if so, why was there not one case in Spain, over which the north wind had passed, not one in Morocco, whence comes the south wind, not one in the islands of the Mediterranean Sea, nor in the countries which are nearest to it on the east and the west? Still more, if we take into consideration the manner in which yellow fever stations itself, ravaging for example, a locality, and yet sparing places immediately adjoining, we are obliged to reject the hypothesis in which ignorance has tried to take shelter. Here, in a few words, is an account of the occurrences observed at Gibraltar. You know the geographical position of the place. Gibraltar is seated upon a rock, which terrestrial convulsions of an antediluvian era have separated by a strait from Africa, and is connected with Spain only by a slip of sandy soil, called the "neutral ground." The particular locality where yellow fever raged with greatest fury was that designated the "sea-gate," beyond which lies the neutral ground, where the pestilence stopped short. The population emigrated to that narrow sandy flat, and there established itself in tents, at a pistol shot from the town. The emigrants, who never entered the town, had not one case of yellow fever among them; yet, they were so near the hot-bed of the disease, in such close proximity to the ditches of the fortress, that they could, so to speak, converse with the sick shut up within its walls. Does not this fact absolutely demonstrate, that the winds have no influence whatever in propagating, nor, *à fortiori*, in causing the outbreak of an epidemic?

The remarks which I have been making apply equally to infectious and contagious germs. The difference between the two I consider to be this:—The infectious germs, engendered under unknown influences produce certain effects in the individuals who receive them: but there the effects stop—the germs die within the organisms which they infected. Originally engendered also under influences which equally elude us, the contagious germs develop themselves, and fructify within the organism which has received them. The contagious germ is, so to speak, *conceived* as the infant is conceived in its mother's womb: but more than this occurs—the germ assimilates the entire substance of the economy—*totus homo morbus fit*—the man who has received the contagion becomes a new centre of morbific emanations.

Gentlemen, Van Swieten, in his Commentaries on the Aphorisms

of Boerhaave, a book filled with many good things, gives his opinion on the matter now before us. In several places, particularly in treating of small-pox and gout, he speaks of the *materia morbosa*. In his chapter on gout, you will find the following passage :—

"Certe, videmus toties in morbis aliquid, non nisi effectis suis in corpore humano cognitum, turbare totum corpus, et assimilare in suam naturam humores antea sanos : qui humores sic mutati constituunt materiam morbosam dictam medicis, et quæ materies morbosa potentiam sæpe habet propagandi eundem morbum. In dysentericis putridum miasma recipitur ab adstantibus et quamvis illud infinite parvum fuerit, omnes humores hominis sani in tubum dysentericum convertit. Parvo vulnusculo cutaneo tantum, applicatur filum pure varioloso imbutum : susceptum illud contagium silet per plures dies, dein febrem accendit, totum corpus turbat, et convertit humores sanos in suam indolem ita ut quandoque numerosæ pustulæ, omnes pure contagioso plenæ, per omnem corporis superficiem nascantur."

This *something*, which reveals itself only in the effects which it produces, this putrid miasm, this morbific matter, is not perhaps any better known to us than it was to the medical observers of old times ; although recently one of our most distinguished men of science believes that he has demonstrated its existence. According to Professor Ch. Robin, morbid germs are formed by bodies holding a first place both in respect of their material importance and their properties. These bodies are the coagulable compounds called *organic substances*, natural animal and vegetable substances, formed both accidentally and artificially.

I am sure, Gentlemen, that you will be pleased by my quoting some of the views on this subject which have been enunciated by Professor Ch. Robin. He says :—

"Whether solid or liquid, or whether suspended in the vapour of water, these organic substances present this peculiarity, that when they become altered, they transmit by simple contact to healthy organic substances, the kind of alteration which they have undergone, or a similar kind of alteration. For the accomplishment of this, it is not necessary that the quantity of the altered organic substance offer a determinate relation in bulk to the substances modified by them ; as is requisite in the chemical actions exerted on one another by crystallizable compounds. Organic substances, the alteration of which has begun under certain conditions of temperature, moisture

&c., transmit this state by mere contact, or in consequence of molecular admixture with healthy substances, even when in extremely minute quantity, because the modification proceeds gradually from molecule to molecule."

"It is by altered vegetable and organic substances that there are produced certain epidemic diseases, such as typhus, dysentery, paludal, and other affections termed general diseases. Through the operation of the same cause, and by means of altered organic substances received into the stomach along with beverages and food, arise the majority of maladies similar to those I have just named, in which the entire economy is implicated; or, to speak with more exactness, in which every organ presents disorders of nutrition, and consequently of every function performed by the organs."

"As examples may be cited the typhoid, variolous, and scarlatinous fevers. In the same category may likewise be included the diseases which result from putrid and purulent infection."

"Among these diseases are some which are pre-eminently contagious: others which have not been proved to be contagious: and also a different class of which it may be said that they are not in any degree contagious, so far as existing experience can be accepted as decisive on the point."

"There exist," says Professor Ch. Robin, "peculiar conditions in virtue of which one individual exposed to the action of these organic substances is attacked, while another escapes: while one exhibits the symptoms in the place of his attack, another shows no symptoms till immediately after he has left the locality, or not till after the lapse of some days."[1]

We shall return to this subject.

In conjunction with this ably propounded hypothesis, I must mention M. Pasteur's new theory of fermentation. This eminent man of science has come to the conclusion, from experiments performed with extreme care, that fermentation is dependent upon sporules diffused in the air; and that each kind of sporule, recognisable by certain characters, possesses the property of originating in a particular medium a different species of fermentation. According to him, there are different sporules for the different fermentations—

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[1] Robin (Charles):—*Dictionnaire de Médecine:* dixième édit., 1855: Article, Maladies ou Affections Générales; et 12e, 1866: Article, Substances Organiques. See also, *Gazette des Hôpitaux*, 2nd August, 1856, p. 361.

the alcoholic, lactic, &c. May there not also exist morbific sporules? May we not in this way explain the morbid fermentation spoken of by the older authors? Bearing in mind the researches of Eidvelt of Prague, and those of Reveil and Chalvet into the composition of the atmosphere in the Parisian hospitals, St. Louis, and Necker, I concur with Professor Pasteur in believing that it would be very interesting to institute an extensive examination of this subject, to compare the organised corpuscles disseminated in the atmosphere of the same place at different times, and of different places at the same time. It appears to me that such inquiries would throw light upon the phenomena of contagion, particularly during periods when epidemics are prevailing.

A sporule diffused in the atmosphere can live only in a latent form, like the grains of wheat in the Egyptian tombs. But if, like the latter, you place the sporule in a place suitable for its living, it will then develop itself, multiply at the expense of the elements with which it meets in the favourable medium, and, according to its species, originate the phenomena of the different fermentations. May it not be the same with the sporules of diseases, which, floating free in the atmosphere, may be only waiting for certain favouring circumstances to enable them to reveal their existence, develop themselves, multiply, and produce the supposed morbific fermentation? Has it not been said that pus generates pus? Perhaps there is a pus-sporule to explain purulent infection; and perhaps there is also a dysenteric sporule, and a choleraic sporule. If these sporules could be detected in the atmosphere, the facts relating to contagion would be materially explained. To make that discovery it will be necessary to follow in the track indicated by Professor Pasteur, proceeding by experiments conducted with the same ability and patience which he has shown.

I have pointed out to you the part played by the organic substratum, and by the specific nature of the ferments in the act of fermentation. I ought to state that Dr. Jules Lemaire, an eminent physician, has very recently demonstrated the essential importance of the nature of the medium in the intimate mechanism of fermentation.[1] In opposition to M. Pasteur, who considers that there is a special ferment for each kind of fermentation, M. Jules Lemaire

[1] Lemaire (Jules):—Nouvelles Recherches sur les Ferments et les Fermentations. [Lu à Académie des Sciences, en Septembre et Octobre, 1863.]

makes out that there are neither special microphytes nor microzoa in particular fermentations, and that the existence of one or other is contingent upon the medium. Thus, in a liquid which is neutral or slightly oxidated, and contains organic substances in infusion, microzoa (bacteria and vibriones) appear; and by their aid, fermentation is accomplished. But if the substances are acid, it is then microphytes which are developed, and it is then, by their assistance, that fermentation takes place. But that is not all: in acid substances, fermentation begins with microphytes, and when the acids have to a great extent become transformed, microzoa appear, the smell at the same time becoming extremely fetid: the changes take place in an inverse order when an originally neutral medium becomes acid; that is to say, the appearance of microphytes precedes the appearance of microzoa.

These experiments have only a remote analogy to the much more complex phenomena of the contagion of diseases: I have only brought them under your notice to enable you to appreciate the very great difficulty of the subject. In fermentation, where the whole process is seen, the specific character of the ferments, or of the living agents of fermentation, is recognised: the contagion of diseases, on the other hand, proclaims the unimportance of these agents and the omnipotence of the medium. In the act of contagion, we can scarcely perceive the material agent, and are obliged to prove its existence by induction, or from analogy.

Can morbific germs, infectious or contagious, remain in a latent state external to all organic life? Recollect the epidemics of small-pox on board the Wellington, and the cases of ophthalmia at Gibraltar: remember the woman who died of glanders from having worked among horse-hair from Buenos Ayres. In the same way, a contagious disease desolates a family at a particular period, and then disappears, to reappear, however, after a certain time with equal severity, but independent of any new contagion from without; there being in fact nothing to which the reappearance can be attributed, excepting that the germ of the disease had remained concealed where the family was living, in the hangings of the furniture and of the apartment, just as the variolous germ remained in the structures of the Wellington, as the germ of ophthalmia remained in the barracks of Gibraltar, as the virus of glanders remained in the horse-hair from Buenos Ayres.

A girl of nine years of age was carried off by malignant diphtheria.

On the first manifestation of the symptoms, her two sisters were removed to a distance from the house, and did not take the disease. But eight months afterwards, on returning home, the elder of the two was seized with diphtheria, which invaded the larynx; and I was called in to perform tracheotomy. This child died, as her sister had died, from diphtheritic poisoning. Again, on this occasion, as soon as the disease was recognised, the surviving sister, aged five years, was sent off to the residence of her grandmother; but she carried with her the germ of the malady. Sore throat very soon declared itself, and in seven days, croup necessitated tracheotomy, which was in this case a complete success.

Two circumstances in the history of these children require to be looked at separately; viz. the preservation of the germ, external to the organism; and the incubation of the malady. By the term *incubation*, we must understand the time which elapses from the entrance of the morbific cause into the economy till it manifests itself by producing the symptoms of the disease which it determines. It is probable that the last of the three children received the diphtheritic poison at the same time as her deceased sister, the evolution of the malady being slower in the one case than in the other. The period of incubation, is, at least in some diseases, as you know, longer or shorter in different persons according to their individual peculiarities.

But however long the period of incubation may be, its duration is not indefinite; and if sometimes it appear to be prolonged beyond the ordinary term, there has not really been incubation. The morbific germ had not entered the organism, but had remained on the surface of the external tissues, exactly as in the cases we have now been considering, in which it was preserved in the clothes of a patient, the drapery of an apartment, or the woodwork of a ship. This explanation will be accepted when it is seen, that in epidemics, of small-pox for example, persons living in the very centre of contagion are not all simultaneously seized; but that some are attacked immediately, and others much later, and too late to allow us to believe that the incubation began at the same date; while others again are not attacked for a longer or shorter interval after leaving the centre of contagion.

In considering the question of contagion, it is necessary, not only to bear in mind the element of contagion itself, but also, and even more, the conditions necessary for its action.

There are two factors: one is the morbific germ coming from without, and the other is the economy about to receive it. Here, as in every pathological and physiological act, there is required a stimulus, and also support for that stimulus, which Récamier called the reciprocative power; or, in other words, there is required a special aptitude in the organism to respond to the action of the stimulus. Permit me to return to these points, which I have already glanced at in my lectures on small-pox.

Except by the relation between the stimulus and the support, how are we to explain the occurrences attributed to that which is called *predisposition?* How are we to explain, why an individual may expose himself a hundred times to an icy cold, to sudden changes of temperature, without experiencing the least detriment, whilst the same person will take a severe catarrh, an inflammation of the lungs, or a pleurisy, from having been touched in a hot day by a current of mild air coming in behind him at a half open window. The explanation is this: in the first case, there was a capacity for resistance, and, as we say, *a negation of receptivity;* while in the other case, the economy was—excuse the expression—quite open to receive the disease. It is therefore said with truth, that one does not generally take a pneumonia proportionate to the intensity of the cause, unless there exist a predisposition to the disease.

During the prevalence of what are called the common "medical constitutions," all morbific influences act in the same way, in virtue of the common aptitudes which these "constitutions" have imparted to different individuals—then, causes small and great produce similar effects. During an epidemic of influenza, for example, a current of cool air, and a chill when in a state of copious perspiration, occasion catarrh, which assumes the specific character of the prevailing epidemic. When cholera is epidemic, the slightest indigestion will become the starting point of an attack of cholera. You see, therefore, that both contagious and non-contagious diseases are contracted only when there is a special predisposition of the economy to receive them.

When there is no such predisposition, the morbific germ perishes. There occurs exactly what occurs in respect of the act of reproduction in the animal and vegetable kingdoms, where it is essential that there exist a special fitness in the germs, and a special disposition in the individual who ought to receive them—a condition the nature of which it is often impossible to discover. So it is, that on one

side or the other, there is something wanting which is essential. When fecundation does not take place, although the individual seem to possess the conditions necessary for conception, one cannot in an absolute manner attribute this result to a defect in the germ, and can only say, that in the case there was a want of the necessary aptitude. When, on the other hand, fecundation does not take place, although the germ possesses the necessary aptitude, it cannot be said that there is an incapacity of being fecundated, but only that at that particular time the individual was not in a state suited to the accomplishment of the act. Finally, should fecundation not take place, notwithstanding that there exist both an aptitude of the germ and of the individual, it ought to be said, that the failure is occasioned by special conditions impossible to determine. It is essential that there exist the favourable conditions which belong to the germ, and also those which pertain to the individual who ought to receive the germ, besides favouring circumstances external to both.

A study of generation in plants and animals shows that numerous circumstances occur unfavourable to the accomplishment of reproduction. In certain species in which this is particularly the case, the Creator has given an exceedingly lavish supply of reproductive organs. In hermaphrodite plants, the stamina, whose office it is to furnish the fecundating principle, are much more numerous than the female organs: for a single pistil, an infinite number of organs secrete pollen. In the plants in which the male and female flowers are distinct, the number of male flowers is enormously in excess of the female.

In animals, in fish for example, it is not unusual for the female to deposit a quantity of ova so vast that if all were fecundated, or at least if all were hatched, the rivers would hardly be able to contain the produce.

It is the same in respect of morbific germs. Thank God! when they are sown broad cast among populations, they do not all grow up: if they did, the world would speedily become an immense desert. But because all morbific germs do not prove contagia, we are not entitled to deny that they possess a contagious principle.

Here is what occurs when diseases essentially contagious are epidemic: although I have already narrated the following facts, I must again cite them. The tag-sore, or small-pox of sheep [*clavelée*] broke out in a flock of five hundred sheep: fifty were seized, and remained with the rest of the herd. The diseased sheep lay in the

same fold with the unaffected, and both ate their forage from the same rack: the litter common to the affected and unaffected sheep was soiled by the slime and pus from the former. A month later, fifty other sheep had the disease, and in five or six months, the epizootic malady had ravaged the herd: only fifty sheep were not attacked. There can be no doubt that the virus possessed its special aptitude, since it affected nine tenths of the flock. Why, then, was not the remaining tenth attacked? Why were fifty sheep spared? No one can deny their individual aptitude. The existence of this aptitude may indeed have been thus shown:—of the fifty sheep which escaped the contagion when lying in the same litter with the diseased, eating from the same rack with them, constantly coming into the closest contact, mingling fleeces, soiling their noses with the discharges of the contaminated, one or more, long after the outbreak has terminated, will take the disease, simply from passing along a road which had been traversed by a flock in which there was perhaps only a single case of tag-sore.

Human pathology affords similar examples. We every day see whooping-cough, measles, and scarlatina establishing themselves in a family by attacking one or two of its members; at a later period, after an interval of perhaps some months, the disease reappears, seizing individuals who escaped on the occasion of the first outbreak, though during it they were living in the midst of the contagion. Such is the history of an epidemic of diphtheria of which I have already spoken to you, as well as of epidemics of small-pox. In these cases, I again repeat, the seizures occur at so great an interval after the first exposure of the individuals as to make it impossible for us to suppose that the disease was during all that period in a state of incubation.

When persons in the first instance escape, but do not ultimately resist the influence of the morbific cause, it is because they had at first a power of resistance, an absence of receptivity; that is to say, they did not till a later period possess that predisposition which is necessary for the reception and conception of the morbific germ. Some females conceive in consequence of the least possible amount of connection with the male; while others, after having had many times unfruitful connection, conceive by the same male on some particular occasion, there being no apparent difference in the conditions under which the fruitful and the unfruitful intercourse occurred. What happens in respect of persons, happens also in respect of diseases.

You may unsuccessfully on two or three different occasions inoculate a person with a virus, the vaccine virus for instance, and, on making a fourth trial, employing virus obtained under conditions exactly similar to those in which that was taken which you used in the three unsuccessful attempts, you may see the vaccinia develop itself, in one whom you were inclined to believe was devoid of aptitude to receive it.

The remarks now made respecting contagious, are equally applicable to infectious diseases; it matters little whether the morbific germ is developed under the influence of particular telluric conditions, such as marsh miasma, or whether it has been conceived by an animal, as is the case with glanders, malignant pustule, or small-pox: in both classes of cases, there must be a suitable relation between the stimulus and the support with which it meets.

Infection and contagion, then, do not take place proportionately to the quantity of the morbific germ, as some physicians profess to believe. As Professor Charles Robin has told you, quality is paramount over quantity; but it is still more important to take into account the aptitude of the germ, and the aptitude of the receiving organism. Not only is quantity of small consequence; but the history of generation in animals would actually seem to show that the active power of germs is in a ratio inverse to quantity, or at least, inverse to the degree of concentration of the principles which constitute them.

Here again, let me borrow my analogies from Spallanzani. Passionately devoted to the study of the wonders of nature, proceeding in the path of discovery unencumbered by preconceived ideas, happy, as he advanced, to find difficulties which stimulated his inquiring genius, the search for a particular truth leading, as he himself said, to the discovery of other truths spontaneously presenting themselves, Spallanzani belonged to that illustrious generation of ingenious attentive observers, which embraced Fontana, Redi, Réaumur, Swammerdam, and Senebier, and which is continued in our day by our great scientific physiologist, Claude Bernard. The perusal of the work of the Italian naturalist carries along the reader, and affords more charming recreation than the most attractive romance. Many of you are acqainted with Spallanzani's works on the subject of generation, and his experiments on artificial fecundation, made not only on the inferior classes of animals, but also on the mammalia.

Spallanzani found that he could fecundate the spawn of the frog and the toad by spreading over it the semen of the male, either by evacuating it by pressing on the abdomen of the animal, or by taking it from the spermatic vesicles; but that the fecundation of the ova of aquatic salamanders could not be accomplished in that manner. He was well aware that in them natural fecundation does not take place after the laying of the ova as in frogs and toads, but within the body of the mother; consequently, he was obliged to have recourse to other means; for he could not, as he himself remarks, fecundate fœtuses after their birth. He repeated his experiments many times, "varying the proceedings in a thousand ways in respect of the quantity of semen employed, and in respect of the manner in which it was applied to the ova, sometimes touching them slightly with it, sometimes gently bathing them in it, and at other times quite soaking them in it; but he was always equally unsuccessful." Discouraged by his unavailing efforts, he was about to discontinue them as hopeless, when it occurred to him that he had forgotten an important circumstance. He recollected that in his experiments on frogs and toads, fecundation was accomplished by bringing the semen into contact with the ova immediately upon their being discharged from the cloaca. The male, coupled to the female, holds her in a close embrace, so that their posterior parts are kept in contact. In the salamander, copulation proceeds on another plan, the ova being fecundated whilst they are within the body of the female; whereas in the frog and toad the ova are external to the female during fecundation. This condition of distance, Spallanzani had lost sight of. During copulation, the male salamander so places himself that the lower part of his head touches the upper part of the head of the female, their bodies forming an angle, the apex of which is constituted by the union of the two heads; or else, the male and female place themselves nose to nose, in such a way as to have their bodies in close proximity, forming, however, a very acute angle. The male then shakes himself about and squirts a copious jet of seminal fluid from his anal orifice, which, mingling with the water becomes greatly diluted, and in that state reaches, and enters, the anus of the female. Bearing in mind this peculiarity, Spallanzani resumed his experiments. Suspecting that the pure semen was not in a state fitted to produce fecundation, and that its dilution with water was an essential condition, he caused salamanders to discharge their ova by pressing them on the abdomen with his

fingers: he then placed the eggs in water in which he had dissolved a small quantity of semen: of twenty-seven eggs so treated, seventeen became developed.

The failures, therefore, had not in this case depended upon a deficiency in the quantity of the germ. Now, what is true of physiological germs, may be said likewise of contagious and infectious morbific germs. I do not mean to say, that we are entitled to conclude from the facts now stated, that the active power of germs is in an inverse ratio to their quantity: I only conclude that we must take more or less into account the condition of quantity: quality is the condition of principal importance. This statement, I now reiterate, although I insisted upon it when lecturing upon specific influence.

Thus, Gentlemen, it appears, that quantity of germ, but still more quality of germ, aptitude of the individual by whom the germ ought to be received and conceived, and the relative circumstances in which the individual is placed, are the conditions which influence contagion and infection.

These conditions as I have said are far from being always met with: upon this point, experience has given a distinct verdict. Some persons possess an absolute power of resistance: there are individuals who pass unharmed through every kind of epidemic, be it influenza or cholera, scarlatina or measles, small-pox or dothinenteria, typhus or yellow fever: there are individuals whom it is impossible to affect with the vaccine virus—inoculate them twenty times, and you will obtain no result: in them, if I may use the expression, the soil is barren—in it the seed cannot germinate. There are others again, in whom the power of resistance is only temporary. It is, in general, difficult to find out the conditions upon which this power of resistance depends: in some cases, however, they can be got at, though we can never become intimately acquainted with them.

Every farmer will tell you that pregnant ewes are less liable than other sheep to contract contagious diseases; but that as soon as they have brought forth their young, they return to a state of liability similar to that of other sheep. The same remark is to a certain extent applicable to women. Magendie explained this fact by saying that the sanguineous plethora, which is usually more or less decided in pregnant women renders absorption more difficult; and that after parturition, it again becomes more easy, in conse-

quence of the plethora being diminished by depletion of the vascular system, and by the comparative emptying of the abdomen caused by the decrease in the volume of the uterus, so that after parturition women and female animals resume their aptitude to receive the germs of contagious diseases. That is the physiological explanation. It is not for me to discuss it. It is easy to understand why it should be accepted: but it matters little whether it be received or rejected, as the fact will still remain.

It has been alleged that great overflowings of the heart, such as arise from emanations of joy and maternal love, fortify the system against contagion, while depressing moral emotions, such as fear, increase its susceptibility.

It is known that the ability to resist contagion varies with the age of the individual; there is less power of resistance in the youth than in the old man; and, all other conditions except age being equal, old men resist contagion better than adults.

Again, it is well known that an anterior contamination generally confers an absolute immunity from any subsequent contamination. In respect of small-pox, this is the case with very few exceptions. Though instances do occur of persons contracting indurated chancres several years after a first attack: though, consequently, there are examples of second attacks of syphilis similar to those published by Dr. Follin and other conscientious observers of unquestionable credit, such examples are rare, and do not controvert in the least degree the law of immunity as enunciated by Dr. Ricord. In fact, the statement applies to syphilis in the same manner that it applies to small-pox, measles, scarlatina, dothinenteria, and yellow fever; that is to say, that while the immunity acquired by a first attack is universally admitted to be the rule, it is also equally admitted that the rule presents a considerable number of exceptions.

I have laid before you accounts of second attacks of small-pox, and you have yourselves seen such cases in the hospitals. You have also seen a well-marked similar occurrence in respect of dothinenteria. Some months ago, the patient to whom I refer occupied bed No. 7 of St. Bernard's ward. She came into hospital with fever, general pains in the limbs, lumbar pains and headache; she complained of sleeplessness. The appearance of the tongue, copious diarrhœa accompanied by gurgling in the right iliac fossa, and finally an eruption of rosy lenticular spots left no room for doubt as to the diagnosis. This woman, however, said that, four years previously,

she had had precisely similar symptoms. At that time she was attended by my honourable and accomplished colleague Professor Rostan, in whose wards she remained for four months. The duration of the illness enables us to come to a probably correct conclusion as to its nature; but the circumstance which removes all doubt on the point is the patient distinctly recollecting to have heard it stated by those around her at the time, that she had "typhoid fever." I have in my private practice met with an example of a person twice taking this disease. A girl, twelve years old, took dothinenteria: the case was very severe, and the illness lasted fifty-seven days. In the following year, she had another serious attack of the same disease. The symptoms were quite as distinctively characteristic as on the first occasion; and the duration of the disease was fifty days.

Whooping-cough, which generally confers immunity for the future, may likewise occur more than once in the same subject. A girl of three and a half years of age, whom I had attended ten months previously for this disease, again came under my care with whooping-cough, of which she had a second attack as severe as the first.

These exceptional facts do not at all invalidate the general rule, that one attack of a contagious disease generally protects the individual from it for the future. It would appear that the virus or morbific matter, upon its entering the economy for the first time puts in motion all therein that is fermentable [*tout ce qu'il peut y avoir de fermentescible*], and so thoroughly destroys it, that the leaven—the contagion—when introduced again, finds nothing whereupon to exert its action.

A similar immunity from the virus of contagious diseases is conferred by habitual exposure [*accoutumance*]; and also, immunity from infectious germs is bestowed by acclimation. In respect of both, however, the immunity is more apparent than real. A European, for example, comes into a region where yellow fever is endemic: should he have the good fortune to sojourn there for a certain time without there being an epidemic of the disease, he will have acquired such immunity by his residence, that when the fever breaks out, his immunity will be equal to that of the indigenous inhabitants. This is what is alleged by those who hold that immunity is derived from acclimation. According to them, it is well known that the native inhabitants of a country enjoy so great a

degree of immunity that even when they remove to another climate they may come back to their own locality without incurring any risk from contagion, although it might be supposed that during their absence they had lost part of their power of resisting it.

The same remarks apply to marsh fevers. At our stations on the Senegal where our troops, when they penetrate inland, are cruelly decimated by terrible attacks of pestilential fever, the indigenous negroes suffer very little in this way. So it is in our Algerian possessions: although very few Europeans escape the African fevers, the Arabs suffer less from them although they are not original inhabitants of the country: like our colonists and soldiers, they have emigrated to it, but having dwelt in it for seven or eight hundred years, the race has become acclimated. In the case of the Europeans, on the other hand, there has not yet been time for acclimation, for they have only been in Algiers since the conquest.

It would appear then that acclimation in a certain number of cases confers absolute immunity from marsh fevers; but that in other cases the immunity is only relative. The Arabs themselves take the disease, though in a less degree than Europeans. In the departments of France forming the old province of Sologne, where fevers always prevail, the inhabitants pay them a heavy tribute, as appears from the statistics drawn up by commissions of the recruiting department. Sologne, indeed, is never able to furnish its proper annual contingent to the conscription, so small is the number of its really efficient men: nearly the whole population has a constitution deteriorated by the infectious miasmata to the influence of which they have been more or less subjected. Many have a bistre colour of the skin, with engorged spleen and liver, the characteristics of marsh cachexia. To this fact, so opposed to their opinions, the reply made by the believers in immunity acquired by acclimation is, that the Solognese enjoy a *relative* immunity. They say, let a Solognese and a Parisian go to live in a place where these fevers are at the time prevailing: the first will take a tertian or quartan ague, while the second will take a pernicious fever. When we come to consider the subject of marsh fever, we shall see, that the immunity about which I have now been speaking appears to belong to certain races, while there are other races which do not possess it, and are incapable of being acclimated.

In respect of a contagious virus, it is not a question of immunity

produced by acclimation, but by habitual exposure [*accoutumance*]. This is the explanation given of the fact, that nursing sisters and physicians can live in the midst of contagious diseases without contracting them. This fact has been compared with that immunity from poisoning by arsenic and opium respectively acquired by arsenic eaters and opium smokers—a new version of the more apocryphal story of King Mithridates. Poisonous doses of the most dangerous substances may be taken without harm by persons who have been long accustomed to their use in small doses.

The alleged facts now mentioned in respect of acclimation and habitual exposure have not been, in my opinion, by any means demonstrated; and there are many other facts which are contradictory to them. From among the latter, I shall only cite one example. During the Crimean war, typhus made a relatively larger number of victims among the medical men than among the soldiers. Yet the medical men were placed in the conditions alleged to confer immunity, for, from the beginning of the epidemic, they were in contact with the sick.

If there be any ground whatever for holding the opinion which I am now combating, it is, as I have just said, that the immunity spoken of is more apparent than real, except in some exceptional cases. This does not arise from the individuals possessing a natural capacity to resist morbid influences, but upon their having acquired such a capacity at the cost of an attack of the disease, of which there remained neither trace nor recollection, or of which the characteristic symptoms had been mistaken. Having spoken to you of *variola sine variolis*, of measles without eruption, and of "defaced" scarlatina [*scarlatine fruste*], you can understand that attacks of these exceedingly contagious diseases, by passing unnoticed, though conferring immunity from subsequent attacks, make it appear as if certain individuals were originally exempt from the risk of contagion, whereas their exemption has been acquired by their having had the disease in question.

Let me state the facts. Drs. Chervin, Louis, and I were sent to Spain to study yellow fever, when it was prevailing as an epidemic at Gibraltar.[1] You know with what rigorous precision my honourable colleague Dr. Louis was in the habit of observing patients and

[1] Chervin, Louis, Trousseau.—Documents recueillis par la Commission Médicale Française envoyée à Gibraltar. Paris, 1830.

drawing up the reports of their cases: nothing could escape him. To enable him to draw up his statistics satisfactorily, he wished to see the whole population. This was easily accomplished at Gibraltar, where the inhabitants are few. We therefore saw everybody; making diligent inquiry at the same time for those who had had yellow fever in the previous epidemics of 1804 and 1813. Upon investigating the question, whether a previous attack had conferred the immunity which many seemed to enjoy, we found that among those who took the fever in 1828, there were only twenty-four persons who had previously had it. It is a remarkable fact that in reference to some of those whom the scourge spared, we were assured by persons who spoke from personal observation, that they had "imbibed a former epidemic with their mother's milk," having had a mild attack of yellow fever which had lasted three or four days. Similarly mild cases we ourselves observed in the epidemic of 1828. For some days, the patients experienced a general feeling of discomfort, which did not prevent them, however, from following their usual occupations. Under such circumstances, it is easy to understand how the disease might remain undiscovered.

In like manner, some persons owe their power to resist vaccination to their having had at some anterior period an exceedingly slight attack of distinct small-pox, characterised by a few pustules to which no attention was paid, or by pustules confined to the arch of the palate, as occurred in a case which I met with; or again, the exemption may be conferred by the individuals having had small-pox during intra-uterine life. However mild, however distinct the attacks may have been, they suffice to confer immunity: they not only render individuals incapable of taking small-pox, but they likewise incapacitate them from taking vaccina.

I am not, however, disposed to deny that there are individuals who possess an absolute immunity. To employ a comparison which I formerly used, I admit that in some individuals the soil is completely barren, and that in others it only enables the seed to germinate badly. On a former occasion, I cited examples to you of subjects upon whom neither exposure to the contagion of small-pox, nor even inoculation produced any effect: I know, also, that there are other individuals, who, although they have never been vaccinated, and have never had small-pox, yet when they do take small-pox, have it in a very modified form, which seems to demonstrate the existence of at least a relative immunity. The point upon

which I wish to insist is that absolute immunity is exceedingly rare.

One word more, Gentlemen, on the *transmission of germs.*

Some germs, such as that of syphilis, are transmitted by *simple contact.* That the contagion take effect, it is sufficient that the venereal virus be in contact with a mucous surface like that of the glans: it is not necessary that there should be any lesion, excoriation, or ulceration, which, however, if present would open a wide door for absorption. An often-repeated experiment has conclusively determined this fact. The experiment to which I refer consists in placing pus taken from a syphilitic ulcer under a watch-glass, and in contact with a healthy mucous membrane: the result is another ulcer—a specific chancre. Malignant pustule is also transmissible by simple contact. Shepherds often become affected by malignant pustule by skinning sheep which have died of *sang de rate:* the disease becomes developed in the eyelids, the cheeks, and other parts where there is no lesion of the integuments. I am aware that it has been said that in these cases the contagious pus had come in contact with some slight abrasion of the skin; but this is a mere supposition, for individuals the most scrupulously careful of their persons, and who have affirmed that they had not the slightest abrasion anywhere, have taken the disease from sheep in the manner I have now described. It must be admitted, however, that transmission by simple contact is the rarest manner in which contagion is transmitted.

The two more common ways are *transmission by inoculation,* and *transmission by inhalation.* In the first case, the virus is introduced into the system by a denuded surface, or by an artificial opening: to the latter mode, the use of the term *inoculation* is more properly restricted. Inoculation is the most certain manner of transmission; for the virus being placed beneath the epidermis by the lancet, or brought into contact with a denuded surface, finds the open mouths of the absorbent vessels, and through them effects an entrance into the organism. The diseases of which I have been speaking, though contagious by simple contact, are in a much greater degree contagious by inoculation.

Take small-pox, measles, and scarlatina. It is unnecessary to insist upon the inoculation of the matter of small-pox. As you are aware, for a long period variolous inoculation was the only means employed for protecting the community from great epidemics of small-pox. At present, we hear nothing said about the inocula-

tion of eruptive fevers through the blood; but nevertheless, I ought to remind you that, although the experiments have often been negative in their results, we must concede an important place to the inoculation of the blood of small-pox patients successfully performed by Luigi Sacco in 1849; and of the blood of persons having measles, likewise successfully performed by Home of Edinburgh in 1758; by Speranza of Milan in 1822; and by Michaël of Katona, an account of whose experiments you will find in the *Gazette Médicale de Paris* for 1843.

Finally, you are aware of the fact, upon which I have already sufficiently insisted, that syphilis may be transmitted by vaccination when the vaccine matter has been taken from a subject in whom syphilis is either active or latent.[1] Dr. Rollet, in a work published in 1861,[2] has reproduced and supported the conclusions formerly arrived at by Dr. Viennois, his pupil.[3] The cases reported by Drs. Rollet and Viennois leave no room for doubt as to the possibility of the transmission of syphilis by vaccination. The two cases of M. Lecoq, military surgeon, still further confirm the statements of the physicians of Lyons, previously shown to be correct by facts adduced by MM. Waller, Gibert, and Hubner. From these data it is apparent, that under certain conditions, healthy individuals inoculated with the blood of persons affected with syphilis become affected with syphilis, which first shows itself by a chancre variable in form, and having a special form of induration. It is called by Dr. Rollet the *vaccino-syphilitic chancre*.

In the second mode of transmission—*transmission by inhalation*—contagion takes place by the absorption of a virus or a miasm by the mucous surface of the respiratory passages, and, possibly also, by simple contact therewith. Here I must pause in my description, that I may make some explanatory remarks.

This manner of transmission has been confounded with infection,

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[1] See Vol. II, p. 124, of this translation: see also a communication made by Professor Trousseau to the Academy of Medicine in 1865, "*De la Syphilis Vaccinale.*"

[2] Rollet:—Recherches Expérimentales et Cliniques sur la Syphilis: Paris, 1861.

[3] Viennois:—Recherches sur le Chancre Primitif, et les Accidents Consécutifs produits par la Contagion de la Syphilis Secondaire. [*Thèse*]; Paris, 1860.

but it much more nearly approaches transmission by direct contact, if indeed it be not identical with it. To explain the spread of certain diseases evidently contagious, it has been said, that the air is vitiated by effluvia from the sick, and has thus become infectious. In the ward of an hospital, containing patients both with scarlatina and small-pox, other patients, occupying beds far removed from the latter, have taken scarlatina: it has then been said that the original scarlatina patients vitiated the air, that the second class of patients were infected by breathing the vitiated air—in the same way, for example, that individuals are infected, and take typhus, in the ambulances of armies. I do not think that this doctrine will bear the slightest examination. The air is not vitiated: it is simply contaminated. It serves in such a case only as the vehicle by which are transmitted the volatile emanations from variolous and scarlatinous patients: it is not vitiated any more than is the pus of a bubo serving as the vehicle of the germ of syphilis. Air and pus have each their own physical and chemical properties: and in addition, the most delicate analysis and the best microscope can detect nothing more. The contaminated air serves as the vehicle for the virus of small-pox, just as the scabs from the pustules were in former times pounded down and dusted over the bread-and-butter intended for children subjected to inoculation, or introduced according to the Chinese fashion into the nostrils; or, as threads soaked in variolous pus were used by early inoculators. Though in these cases, the contagion is transmitted in a more direct or at least in a more palpable manner, the transmission is similarly affected, when, by inhalation, the morbid principles transported in the air, come into contact with the nasal fossæ and the bronchial tubes, penetrating to the remotest ramifications of the respiratory apparatus. Nevertheless, the third mode of transmission—that by inhalation—has been distinguished from the two others.

Contagion, whether it be mediate or direct, is not infection. Both may originate in telluric or atmospheric influences; but there is, I repeat, this essential difference between them, that contagion transmits to a person in health morbid germs which have been developed in a diseased person, while this is not the case in respect of infection.

In conclusion, let me recall to your recollection a fact which I have just mentioned, to the effect that contagious diseases in passing

from one to another animal species lose their power of transmission: such is the case with hydrophobia. There are other contagious diseases which change their form in transmission. I dwelt on this fact at so much length when speaking to you of vaccina, *eaux aux jambes,* and cow-pox, *sang de rate, charbon,* and malignant pustule, that I need not resume the discussion.[1]

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[1] See page 98 and following pages of Vol. II of this translation.

LECTURE XXIV.

OZÆNA.

A very Common Affection.—Must not be confounded with Fœtor of the Mouth or Throat.—Fœtor of Ozæna is altogether Peculiar.—Sometimes Dependent on Alteration of the Secretions.—Fœtor of Inflammatory Secretions in some persons.—Constitutional Ozæna.—Symptoms.—Syphilitic Ozæna very frequent.—Ulceration of the Mucous Membrane: Necrosis.—Diseases of the Maxillary Sinus.—Topical Treatment is the most usual.—Constitutional Treatment is very useful in Syphilitic Ozæna: also of considerable benefit in Herpetic and Scrofulous Ozæna.—Powder for snuffing up the Nose.—Injections.—Treatment must be very patient and very varied.

Gentlemen:—You have repeatedly seen patients with ozæna [*ozène, punaisie*] in the clinical wards; and on several occasions I have directed your attention to the different causes of this cruel affection. I very recently showed you a young girl who has had from infancy ozæna which I considered to be herpetic; and almost at the same time, I had under treatment in the male wards a patient with syphilitic ozæna.

Whenever the nasal secretions become fœtid, we say that the patient has ozæna; but the causes of the fœtor are so different, and the proper treatment so varied, that I cannot allow the occasion to pass without taking a short general view of the question.

The horrible fœtor of the breath which constitutes that which we call bug-stench [*punaisie*] is an affection so disgusting, and yet unfortunately so common, that you ought from the very beginning of your career to be acquainted with its causes and treatment.

First of all, Gentlemen, it is important not to confound the ozæna which proceeds from the nasal fossæ with fœtor of the breath caused by an affection of the mouth and throat. In persons who have had frequent attacks of inflammatory sore throat there often remains submucous fistulæ which secrete fœtid pus, and wherein

accumulate some of these sebacious products, so often seen in the furrows of the tonsils, and which are ejected in the form of small whitish, cheesy concretions, which when crushed emit an intolerable stench. It is unnecessary for me to remind you of what takes place in cancerous affections of the pharynx, larynx, and upper part of the œsophagus.

In persons whose breath is most free from taint, the normal secretion of the mucous membrane of the mouth, after accumulating during the night on the tongue and teeth, acquires a disagreeable odour. If there be an inflamed condition of the gums and mouth, the secretion becomes more abundant and more fœtid, and unless the requirements of the toilet are carefully carried out, this disagreeable state continues till the secretion is carried downwards at a repast. But should the individual have carious teeth, suppuration in the centre of the caries, or around the diseased teeth, often occasions a fœtor which cannot be got rid of, however great may be the attention given to the mouth.

Let me also remark that in some individuals the secretions of the mouth are naturally fœtid, and incapable of being rendered otherwise by the most rigorous cleanliness. I need not remind you of an analogous condition of the feet, ears, and axillæ.

What I have now said will I think suffice to prevent you from falling into any confusion. It is important to avoid mistaking that fœtor of the breath which proceeds from an affection of the throat or mouth for that which originates in the nasal fossæ; but it is equally important to avoid the opposite error. Such mistakes, however, are always easily avoided. The simplest means of arriving at a correct diagnosis is to ask the patient to shut alternately the nose and mouth during expiration: when this is done, there is no difficulty in recognizing the source of the fœtor. I ought, however, to add that the specific bad smell is chiefly met with in that form of ozæna called constitutional, and which is peculiarly allied to the scrofulous or herpetic diathesis.

The two cases at present in the clinical wards give you a sufficiently correct idea of the nature of the fœtor met with in the different kinds of ozæna. In the young girl who has suffered from this disease from infancy, there is something in the smell which excites sickness: in the other patient, who is suffering from constitutional syphilis, the fœtor is no doubt very great, but it is less nauseating.

I shall not dwell longer, Gentlemen, upon details the value of

which you will be better able to appreciate at a more advanced period of your studies.

Persons attacked with ozæna fortunately possess the privilege of not perceiving the bad smell, except in rare exceptional cases, as, for example, when the maxillary sinus is alone affected. The very disease of the mucous membrane which produces the ozæna destroys the sense of smell. It consequently happens that the affected individuals, without being aware of it, are frequently frightful sources of misery to those around them, who sometimes, from politeness or pity, conceal their disgust.

Sufferers from ozæna become incapable of distinguishing between good and bad odours; and at the same time lose the sense of taste, or, to speak more correctly, that portion of it which is associated with the sense of smell.

I need not remind you, Gentlemen, of the fact stated in all your books on physiology, that certain flavours are perceived by the smell, whilst most flavours are either not perceived at all, or only to a slight degree, when the nostrils are closed, or when the sense of smell is lost. Put lemon juice into one glass, and into another water acidulated with acetic, sulphuric, hydrochloric, or other acid, and you will find it impossible to distinguish the taste of lemon juice from that of the other acid liquids, if you hold your nose in such a way as to close the nostrils.

All secretions in contact with the atmosphere, unless renewed, become altered in composition. This alteration is more considerable in some persons than in others, in virtue of conditions which I find it rather difficult to describe, but which perhaps belong as much to the quality of the secretion at the time of its formation, as to the special state of the secreting organ. In some persons the nasal secretions, like the pharyngeal, vaginal, and anal secretions, undergo rapid change, and acquire an excessive fœtor, not perceptible in other individuals much less particular in the observances of the toilet.

Sometimes ozæna is solely dependent upon the odour of the altered nasal secretion. When the mucous accumulation is removed from the nostrils in such cases, the breath becomes quite pure; but after some hours, the fœtor returns, if the mucous secretions have been allowed to reaccumulate in the nasal fossæ. It is obvious, that the remedy for an infirmity of this kind consists in blowing the nose frequently, and keeping it very clean.

It appears then that in some persons it is normal for the secretions of the mucous membranes, like those of the skin, to be characterised by fœtor. In such individuals, when the mucous membranes or the skin are affected by acute or chronic inflammation, this normal fœtor becomes very greatly increased. You know how easily, particularly in fat persons, a bad smell is produced by chafing under the mammæ, in the folds of the thighs, or around the anus; and that sometimes there is no preventing this by the most scrupulous attention to cleanliness. So it is, as you know, in inflammatory affections of mucous membranes; for you must have been often struck with the fœtor of gonorrhœal matter in some individuals. The fœtor lasts as long as the acute stage of the inflammation; and in some persons, even after the inflammation has passed into the chronic stage, the inflammatory secretions continue to emit an intolerable smell, however brief may be the time during which they are allowed to remain in the situation in which they were secreted. If the inflammation of the mucous membrane is of a special character, the secretion may be fœtid from the very first moment of its formation.

It is necessary, Gentlemen, to enter into these details, to enable you to understand the history of ozæna. There are many persons, who, when they have coryza, discharge mucous secretions possessing an exceedingly disagreeable odour: it is not, however, the odour of constitutional, but of what may be called the first stage of accidental ozæna. Should the coryza become chronic, the secretion will undergo change whilst remaining in the nasal fossæ, and the fœtor may resemble that which is met with in certain specific inflammatory affections of the pituitary mucous membrane.

The form of the disease designated *constitutional*, a term, however, which I do not justify, is not in general met with till after the years of childhood, even when there have existed from birth some of these anatomical lesions of which I shall afterwards speak, and which almost invariably lead to ozæna. The malady seldom begins to make its appearance in subjects under four or five years of age; but towards puberty, it assumes considerable proportions, and continues considerable during adult years, decreasing but not entirely disappearing at a more advanced period of life. This form of ozæna is characterised by a repulsive sickly smell, bearing no resemblance to any other smell: generally, the nasal secretions are purulent, sometimes they desiccate, forming crusts moulded in the

passages, and when this is the case, they are almost always mixed with a little blood, if an effort has been required to expel them. There is often a very abundant purulent discharge; and it is not in such instances that the stench is most disagreeable, unless the ozæna proceed from disease of the maxillary sinus, from which, the pus having therein accumulated, may come in gushes, consequent upon certain movements of the patient. Upon examining the interior of the nasal fossæ by the aid of a small speculum, some redness of the mucous membrane is nearly always found.

Deformity of the nose, from flattening of the root, is pretty frequently observed in ozæna. It has for that reason been assigned as a cause of the disease: it has been supposed that the consequent structure of the nasal fossæ prevented the evacuation of the mucous secretions, which become altered from being long pent up. Bear in mind what occurs in syphilitic ozæna of the adult during the course of which fœtor may exist, and in fact generally does exist, without there being any disease of the bones or deformity of the nasal fossæ: bear in mind also, that in the majority of adults attacked by ozæna there is no deformity of the nose. The natural inference, therefore, is that the flattening of the root of the nose and the ozæna both proceed from the same cause—that is, from chronic inflammation and ulceration of the mucous membrane, with consecutive necrosis of the vomer and some portions of the ethmoid bone.

Moreover, persons are often met with who have nostrils so exceedingly narrow that the air does not pass through them in quantity sufficient for the requirements of respiration, and in whom nevertheless the nasal secretions are always inodorous.

There are other and rarer cases, in which there is no deformity of the root of the nose, in which the nasal secretions present no unusual appearance, and in which neither is there pain in the head nor tension of the upper jaw to indicate a state of acute or chronic inflammation. The mucous membrane in these cases is also without any of the characteristics of inflammatory action.

Again, when there is nothing to lead one to suppose that there is inflammation of the pituitary membrane, or necrosis of the bones, —when the individual attacked by ozæna has the conditions of perfect health—we find ourselves forced to admit that there is a peculiar fœtor of the nasal secretion, like that observed in the feet of some people: this is the form of the disease to which the term *constitutional* ozæna ought to be restricted. To follow out the

comparison:—we should not be justified in confounding the bad smell which proceeds from the feet of those who neglect necessary ablution and have no skin disease, with the disagreeable odour so often observed in the feet of patients suffering from chronic eczema of the feet, and particularly from the sequela of cutaneous inflammation, such as are seen between the toes in the course of venereal diseases.

Alongside of this kind of ozæna, which is really constitutional, we must place that other form of the disease which depends upon the herpetic diathesis, and which is usually seen along with the ophthalmia called scrofulous, and swelling of the upper lip. It must not be supposed that every herpetic affection of the mucous membrane of the nasal fossæ gives rise to ozæna any more than that herpetic affections of certain parts of the body are necessarily accompanied by fœtor: but just as eczema of the feet and vulva produce secretions of most disgusting odour in some persons, so does chronic eczema of the mucous membrane of the nasal fossæ produce in some patients a secretion emitting a most revolting smell.

The most frequent cause of ozæna is undoubtedly syphilis. When the system is contaminated by the venereal disease, coryza is very common, and although, in the majority of cases, it does not cause fœtor of the breath, yet it gives rise to it in the same way as do herpes and scrofula in some persons. But however great the fœtor may be in such cases, it never equals that of constitutional ozæna. Syphilitic ozæna gives rise to ulceration and necrosis, and is the severest form of the disease.

A membrane so delicate as the pituitary membrane cannot long with impunity be the seat of inflammation: ulcerations frequently follow; and Dr. Cazenave of Bordeaux, to whom we are indebted for interesting researches on the subject now before us, has seen ulcerations even on the floor of the nasal fossæ: by using a speculum, similar to that employed for the exploration of the auditory passage, ulcerations can easily be detected upon the septum and those parts of the nasal passages nearest to the opening of the nostrils. These ulcerations become a new cause of ozæna in a way which I shall now explain.

Whatever may be the cause of the ulceration, the sub-mucous cellular tissue is easily invaded by it, and the bone itself soon becomes affected. From the very first moment of the existence of this lesion, it becomes a new cause of ozæna; and even when the original disease

is quite cured, the fœtor continues till the necrosed portion of the bone has exfoliated, or been removed by surgical interference.

Although the smell is much less horrible when there is no necrosis, the infirmity is still a disgusting one for which patients often seek treatment at our hands.

When the arch of the palate, the nasal process of the superior maxilla, the vomer, and the nasal bones are involved in the necrosis —when there is actual destruction of the bones of the nose—the ichorous suppurative secretion is profuse, and the fœtor is shocking, although it does not possess the peculiar odour of constitutional ozæna.

Your surgical professors have taught you that the necrosis which follows gunshot wounds, fractures of the bones of the face, and sometimes even that which depends on the existence of polypi, may produce ozæna. But diseases of the maxillary antrum are still more frequent causes. I was recently consulted by a man of forty years of age, who, with the exception of the affection now under examination, was in good health: he complained of ozæna, which he said had been for a long time the torment of his life. He was standing: I caused him to throw his head backwards, and shut his mouth, so as to be compelled to breathe through the nostrils: to my surprise, I could detect no fœtor in the breath. He then told me that he could produce the disgusting smell at pleasure; and sitting down, with the head inclined very much downwards, he discharged into his pocket-handkerchief a large quantity of pus, which exhaled an intolerable stench in my consulting room.

I have, Gentlemen, but imperfectly sketched the picture of ozæna. I have only attempted to give you a summary view of a common disease, which is of a rebellious character, and not very well understood: I am now particularly anxious to point out to you some of the therapeutic means by which we sometimes cure, and often palliate this cruel infirmity.

First of all, let it be distinctly understood, that we can do nothing, or next to nothing, for ozæna dependent on necrosis: it is only too evident that we can exercise no control over such a disease: the dead bone may become detached in whole or in part, and yet the odour remain as long as there remains a fragment of necrosed bone. You only require to glance at the skeleton of the nasal fossæ to form an idea of the difficulty of expelling some of the portions of the dead bone. When the necrosis is very extensive, the ozæna may last for

a long series of years, surgery generally being unable to afford any relief.

At the end of May 1863, I saw at the Hôtel du Louvre, in consultation with my honourable colleagues Drs. Higgings and Shrimpton a young English officer of the Indian army, who had for a long time been suffering from syphilitic ozæna. He had had, on the preceding evening, a sudden and terrible suffocative attack, caused by the presence in the posterior nares of a foreign body, which had subsequently fallen into the throat. In the midst of his suffocative convulsions, he seized with his fingers, and finally drew forth a large irregularly shaped and rough-edged piece—about a fourth part—of the ethmoid bone. On the same day, cerebral symptoms supervened, under which he died within twenty-four hours. It is probable that there was suppuration of the meninges of the brain, at the points corresponding to the cribriform plate of the ethmoid bone. You perceive, Gentlemen, that when there exists necrosis of this description, expulsion of the dead bone is almost impossible, and exfoliation can only take place in small splinters, and therefore very slowly.

Ulceration or necrosis of the walls of the antrum, or chronic inflammation of the mucous membrane which lines it, will also produce a kind of ozæna for which we can do little. In the majority of such cases, the only means of cure is to make an opening into the antrum, through the superior dental arch, and thereby directly introduce therapeutic agents.

In all cases in which we can direct our treatment to the cause of the inflammation of the pituitary membrane, and in which there is no affection of the bones, the cure is easy: thus, for example, in syphilitic coryza without ulceration, mercurials, and the iodide of potassium are generally efficacious, in the same way that they cure chronic syphilitic inflammations of the pharynx and larynx: but when the ozæna is herpetic, we have no longer specific remedies as in syphilis, and then the cure is often unattainable. Some slight benefit may be derived from preparations of arsenic, iodine, and sulphur; but it is upon topical treatment that we must principally rely. It is still more difficult to obtain favourable results from treatment, when we have to contend against the scrofulous diathesis; and although we may to some extent modify the state of the system by placing the patient under good hygienic conditions, and giving him certain medicines, (the triviality and insufficiency of which you

know,) it is necessary to trust almost exclusively to remedies which can be applied in a direct manner to the diseased mucous membrane. It is, therefore, upon the topical treatment, that I am now going chiefly to insist: it is the kind of treatment which will render you the most signal services.

Powders inspired by the nose (as snuff is taken), the direct application of caustic to the ulcerated parts, and injections of various kinds are the means generally employed; and as they are those which have proved most useful in my practice, I feel that I am entitled to recommend them. Do what you may, it is not easy to accomplish a cure, nor can you ever obtain a cure within a short period. Still, though the means generally employed are imperfect, and not so efficacious as we should desire, we can achieve relatively good results upon which to congratulate ourselves.

There are four powders which I chiefly make use of: I shall now give you the formulæ by which to prepare them.

No. I.	Subnitrate of bismuth Venetian talc	} of each 15 grammes. [232 grains.]
No. II.	Carbonate of potash Sugar in fine powder	2 grammes. [30 grains.] 15 „ [232 grains.]
No. III.	White precipitate Sugar in fine powder	25 centigrammes. [$3\frac{5}{6}$ grains.] 15 grammes. [232 grains.]
No. IV.	Red precipitate Sugar in fine powder	25 centigrammes. [$3\frac{5}{6}$ grains.] 15 grammes. [232 grains.]

No topical application can be of the least use, unless, before its employment, the nasal fossæ have been cleansed by the patient causing cold or tepid water to pass through them. Before the topical medication is proceeded with, the mucous accumulations and the crusts which cover the pituitary membrane must be removed.

I at once begin by employing the mercurial powders. I direct the patient to draw up vigorously a pinch of the powder through each nostril, so as to cause it to penetrate into most of the turnings and hollows of the nose. This proceeding ought to be repeated twice or thrice a day, the frequency being regulated by the amount of irritation produced. Generally speaking, practitioners are not sufficiently upon their guard in respect of the powerfully irritant action of white and red precipitate. Both of these agents, so powerfully efficacious in the treatment of chronic ophthalmia, and diseases

of the skin and mucous membranes, are frequently abandoned just because their irritant action has proved greater than had been expected. The remedy is charged with producing a bad effect, for which the physician alone is to blame. You must remember, therefore, Gentlemen, when you prescribe these mercurial powders, to be on the watch for the irritation which they may excite in the nasal fossæ; and you must order only a small number of inspirations of them in the course of a day, likewise directing them to be continued only for a few days.

There will be a tendency to push too far the use of these remedies in ozæna, from the beneficial results which they produce being as rapid as they are unlooked for. It is no exaggeration to say that in some patients the fœtor disappears a few hours after the powder has been snuffed up for the first time: this result is temporary, I admit, but it is positive, however inexplicable it may be. The effect produced at least proves that the mercurial powders possess the power of modifying the condition of the diseased mucous membrane; and at the same time, it invites us to give the preference to the topical employment of mercury in the treatment of ozæna, in the form of powder, in the manner I have just described; or in the liquid form, according to a plan which I shall forthwith mention.

Though it is necessary to be guarded in the use of the mercurial powders, no such caution is required in employing the mixture of bismuth and talc: patients may snuff it up as often, and in as large quantities, as they please. One might say that the bismuth and talc powder was inert, were an opinion to be formed by the slight amount of irritation produced by applying it; but it is one of the remedial agents on which I place most reliance in the treatment of ozæna, and to which I revert more willingly than to others, just because it may be so freely applied without occasioning any bad consequences.

The chlorate of potash, to which Dr. Henri Saint-Arnoult has given a not altogether unmerited reputation, also renders real service: like the mercurial powders, it possesses the great advantage of causing the smell to disappear whilst it is being used. Were the action of this remedy only that of a disinfectant, it would unquestionably deserve to be recommended; but it merits recommendation on another ground, and that is, that like mercury, it modifies the state of the mucous membrane.

You have seen, Gentlemen, with what rapidity topical treatment

seems to have accomplished a cure in the young girl, our patient in St. Bernard's ward. Looking to the results, it might appear that she is already cured, but it is not so; and as I shall tell you immediately, there are few affections in which both the sufferer and his physician require to exercise more patience than ozæna.

In adults, as their obedience to instructions may be counted on, the inspiration of the powders, though insufficient, nevertheless render eminent service; but in children, this method is almost useless, and in them we must employ injections, as the almost only available treatment, whereas, in adults, they are merely the complement of other measures.

The following are the injections which I most frequently have recourse to:

No. I. Eau phagédénique . . 200 grammes. [℥vj and ʒij.]
[Shake the bottle well before using the injection, so that the precipitate may be thoroughly mixed with the fluid.][1]

No. II. Chlorate of potash . . . . 2 grammes.
Distilled water . . . . . 200 „

No. III. Nitrate of silver . . . . . 5 grammes.
Distilled water . . . . . 100 „

No. IV. Sulphate of copper [or zinc] . . 5 grammes.
Distilled water . . . . . 100 „

There is a very important practical remark which I have to make in relation to these injections. The pituitary mucous membrane is much more sensitive than is generally supposed. It is necessary, therefore, on beginning injections, to use very weak solutions. It often happens that a solution of 5 centigrammes [$\frac{3}{4}$ of a grain] of nitrate of silver, sulphate of copper, sulphate of zinc, or corrosive sublimate in 100 grammes [3 fd. oz. and 1 drachm] of distilled water, is not well borne. Let me add, that this extreme sensitiveness quickly disappears, and that very soon a tolerance for a stronger solution is attained. The solution, however, ought never to be very strong, and should always be proportionate to the sensibility of each patient.

The injections require to be used for several days in succession—twice, thrice, or four times a day, after which the powders ought to be resumed: then, by-and-by, the injections ought to be resumed,

[1] Eau phagédénique. See page 629 of Vol. II of this translation.

their number being diminished or increased from day to day, in accordance with the amount of irritation which they excite in the pituitary membrane, and the curative results which are obtained.

In so obstinate an affection as ozæna, it is easy to see that remedial measures must be continued for a long time ; and if the physician, pleased with apparent success, abruptly interrupt the treatment, the disease will at once relapse. Often, we may exercise the greatest patience, and modify our plan of treatment in many ways, without succeeding in obtaining a radical cure.

The proper plan then is at once, uninterruptedly, and repeatedly in succession, to apply the remedies. When the fœtor has been absent for six weeks or two months, the severity of the treatment may be relaxed by reducing the number of the daily nasal inspirations of powder, or injections. Should the improved state of matters continue, the remedies may then be applied only once in two days, afterwards once in three days, and finally, for some months longer, at intervals of four days.

There is another very important practical point to which I wish to direct your attention. At the menstrual periods, there is generally a great increase in the severity of the symptoms, irrespective of treatment. Even when the plan of medication is directed in the best possible manner, the fœtor generally returns somewhat during menstruation. This also occurs under the influence of any cause which excites inflammation of the pituitary membrane. It is a rule which never ought to be deviated from, to carry out the treatment in all its rigour when the patient is in the special conditions I have mentioned. Even when the symptoms of ozæna have been absent for a long time, the practical precept now laid down must not be forgotten.

However beneficially potent the inspirations of the powders and the injections may be, they are not in themselves sufficient even as topical remedies. Dr. Cazenave of Bordeaux long ago insisted on the necessity of applying modifying agents to the surface of the nasal fossæ and other accessible situations, by means of elastic bougies or rigid sounds adapted to the form of the parts, instruments in fact analogous to those employed in treating diseases of the urethra, bladder, and uterus.

Although topical remedies hold the chief place in the treatment of non-syphilitic ozæna, it would be a great mistake to omit general treatment. Cod-liver oil taken for fifteen consecutive days in every

month, and continued for a long time, is sometimes very useful. The tincture of iodine administered over a period of several months, twice or thrice a day at meals, in a dose of from 5 to 20 drops, often produces exceedingly beneficial results in constitutional ozæna.[1] Arsenical preparations, perseveringly continued, as is usual in treating the herpetic diathesis, are still more powerful adjuvants to the topical medication.

It is hardly necessary to say, that in syphilitic ozæna, mercurial preparations and the iodide of potassium take a place in the treatment even more important than local applications.

Necrosis, polypi, and the different diseases of the antrum of the maxilla, being rather within the sphere of the surgeon than of the physician, I shall not here discuss.

Gentlemen, I must not conclude without repeating, that ozæna is one of the most difficult diseases to cure; but that it is also one of those which it is very easy to palliate, provided reliance can be placed on the cleanliness, docility, and patience of the sufferer, and provided also, that there is a similar exercise of patience on the part of the physician.

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[1] The Tincture of Iodine of the French Codex is simply a solution of about one gramme [15½ grains] of iodine in twelve grammes [four fluid drachms] of alcohol.—TRANSLATOR.

LECTURE XXV.

STRIDULOUS LARYNGITIS, OR FALSE CROUP.

Long confounded with Pseudo-membranous Croup.—Differs essentially from that disease in its nature, manner of invasion, progress, and complications.—Croupy [croupale] Cough presents characters very different from those of True Croup.—False Croup is not a dangerous disease; but still, in some very rare cases, it has caused death.—The Prognosis is serious when the laryngeal affection is the forerunner of peri-pneumonic catarrh.—In the majority of cases, the Treatment ought to be Expectant.

Gentlemen:—When lecturing on diphtheria, I intentionally omitted the consideration of the differential diagnosis of true and false croup,[1] because it seemed more in place to speak of it when discussing the latter affection. The manner of establishing the differential diagnosis is a corollary to the remarks which I propose to make to-day on stridulous laryngitis [*laryngite striduleuse*].

A perusal of the work on croup by Home of Edinburgh[2] leaves a conviction on the mind of the reader, that the author was pretty much in that deplorable state of confusion which, at the time he wrote, had been introduced both into the scientific and practical discussion of the subject. It is evident that he describes under the same name two essentially distinct diseases; and that though he may sometimes have had to do with pseudo-membranous laryngitis, the majority of the cases he reports are cases of false croup. The same confusion pervaded all the writings published on croup from the time of Home to the appearance of Bretonneau's treatise on

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[1] For the lectures on diphtheria—see this translation, Volume II, p. 468—617.

[2] Home:—Inquiry into the Nature and Cure of the Croup. Edinburgh 1765.

diphtheria. This confusion even pervaded the memoirs submitted to the *concours* in 1812—even the memoirs of Vieusseux, Jurine, and Albers of Bremen, which were honorably mentioned by the Academy. Royer-Collard, the reporter of the commission appointed to examine these works, did not avoid it; and his report, in other respects remarkably good, shows that when he wrote, the notions of Home were still in the ascendant. Indeed, the light of truth did not shine upon this chaos of opinion till Bretonneau established with marvellous lucidity the characters which distinguish from each other the two diseases, showing them to be essentially different in their nature, lesions, symptoms, and relative gravity. The one, true croup, is almost always fatal, unless the treatment be prompt and proper; but the other, false croup, is not a dangerous disease, save in exceptional cases.

Stridulous laryngitis, or false croup, is a very common affection. Every physician, in the course even of a very short practice, has often been suddenly summoned in haste to children said to be suffering from "croup." Although the matter has now been pretty generally explained, the error I mention is still committed every day. This explains how it is, that some practitioners congratulate themselves upon their treatment of croup, and boast of an amount of success which would astonish us, if we could suppose that the cases cured were undoubtedly cases of pseudo-membranous laryngitis. Persons who allege that they have cured a great many cases of croup with extraordinary rapidity—in a few hours for example—by emetics, blisters, and leeches, or other means, fall into the very confusion of ideas to which I have now been directing your attention: they allow themselves to be deceived by a symptom, to which they have attached too much importance, and of which they have not perceived the real characters: they, in fact, allow themselves to be misled by a cough very improperly called *croupy*. So exclusively has their attention been occupied with this cough, that they have taken no account of the antecedent history, or at least have paid insufficient attention to the progress of the symptoms—they have not examined the pharynx with that minuteness which was necessary to ascertain whether any diphtheritic exudation existed.

Notwithstanding the frequency of stridulous laryngitis, it is a disease which is rarely met with in our hospitals, and I have only had one case in my wards since I occupied the chair of clinical medicine. The reason of this you can quite understand. The

disease is, in the first place, peculiar to infants and children under two years of age; and, save in a few exceptional cases received into the nursery ward, children so young are not admitted into the Hôtel-Dieu, which by rights is exclusively an hospital for adults. In the second place, from the sudden manner in which false croup declares itself, and from the rapidity with which it yields, it is very unusual for children who are attacked by it to be brought to the hospitals. I shall therefore only recapitulate the particulars of the one case which we have had in our wards; but I should be leaving the subject of croup in an unfinished state, were I not to take this opportunity of speaking to you of the differential diagnosis of croup and false croup, for the latter affection, though rare in hospitals, is very common in private practice.

What then are the characteristic symptoms of stridulous laryngitis?

A child between two and five years of age—the age, observe, at which true croup is also most common—is suddenly seized in the middle of the night—say at eleven o'clock, at midnight, or at one in the morning—with a paroxysm of difficult breathing. He wakes up in a start, in a state of considerable febrile excitement: he has a cough, which is hoarse and very frequent, as well as strong and noisy: respiration is panting, short, and accompanied, during inspiration, by a sharp sound—by a shrill, jarring laryngeal whistle. The voice is altered in tone: during the paroxysms, it is altogether gone, and in the intervals, is harsh and hoarse. In true croup, there is very seldom complete loss of voice: this is an important point to which I shall return.

The symptoms are much more urgent than those which characterise the commencement of an attack of laryngeal diphtheria. Sometimes, the dyspnœa and anxiety are as great as in the last stage of pseudo-membranous laryngeal sore throat; the countenance is turgid, and the eyes express profound terror: the character of the cough, voice, and laryngeal whistling are such as to strike terror in families, and frighten even physicians themselves. However, in half-an-hour, an hour, or in two or three hours, the frightful crisis has terminated: the child becomes calm, sleep returns, his pulse is less rapid, and his skin is somewhat moist: when he wakes, his cough is still croupy, but it is more moist, and in the morning, it is still more catarrhal: respiration is less whistling, and the voice has nearly regained its natural tone. The symptoms generally recur for several

nights in succession, but with diminished severity in each following attack. The patient has usually good days, there being an almost total absence of fever, and general discomfort: the cough continues, but is moist, and much less rough.

Upon questioning the relations as to the history of the attack, you are told, that the child went to bed in perfect health, and fell into a tranquil sleep. You will sometimes be informed that the child had been complaining a little for some days prior to the attack: that he had taken cold, but was going about, eating, and playing as usual: that he had retained his cheerfulness, and followed his accustomed routine: in a word, that there was no change whatever in his habits.

If you inspect the throat with the most minute care, you will be unable to see any false membrane. The mucous membrane is sometimes red: the tonsils may be swollen; but on examining the cervical and submaxillary regions, you will find that there is no swelling of the glands.

It is in this sudden manner, and with these symptoms, apparently more alarming than those of croup, that false croup generally declares itself, the disease which you cure, or I ought rather to say which cures itself; for whatever is done, however inopportune and irrational the medical intervention may be, it is seldom capable of rendering the affection dangerous, so little is there in its nature of a serious character.

However, Gentlemen, there are circumstances which limit this favourable prognosis. Stridulous laryngitis supervenes at the onset, and during the course of certain diseases; so that it is obvious, that under certain circumstances, you may have to do with a peculiarly modified affection.

When speaking of eruptive fevers, I called your attention to the fact that it is common, during the invasion period of measles, when the nasal, ocular, and bronchial mucous membranes become affected, to see the larynx become similarly involved; and I also pointed out that in children, during the first two, three, or four days, before the eruption has come out on the skin, all the symptoms of stridulous laryngitis are sometimes met with. In smallpox, which is also generally accompanied by sore throat involving both the pharynx and the larynx, the occurrence of false croup is not unusual, though it is not so common as in measles.

Gentlemen, false croup may be the starting-point of one of the

most serious diseases of childhood, catarrhal pneumonia, capillary catarrh, which, according to my experience, is more formidable than croup itself. I have long ago explained to you my views on this subject, and I shall again do so, when I come to speak of the pneumonia of children.

Here, Gentlemen, I ought to recapitulate the history of the case to which I alluded at the beginning of this lecture. From it you will learn a fact, which I shall afterwards have to state, that tracheotomy may be useful in false croup; and it will likewise show you, that false croup may be the starting-point of fatal pneumonia. The following are the particulars of the case to which I refer.

In January 1863, a female infant was received into my wards. The *interne* on duty observed that she had very great difficulty in breathing, but no suffocative paroxysms. According to the statement of the mother, the dyspnœa had increased rapidly within the last few hours. The child had had cough for some days: upon examining the chest, however, no sign of pleurisy or bronchitis was discovered. Inspiration was very laboured, and somewhat whistling: the cry was hoarse and muffled: the obstacle to respiration was unquestionably situated in the larynx. There was no false membrane in the back part of the mouth, nor had the child thrown off any: still, though it was only a case of false croup, M. Dumontpallier, without hesitation, immediately performed tracheotomy, as it was necessary to prevent suffocation. The operation was easily accomplished; and the patient breathed freely as soon as the tube was fixed. The infant passed a good night; and next morning I found that there were no morbid sounds in the chest. The infant's appearance was good; and she took the breast with satisfaction.

On the third day after the operation, an attempt was made to withdraw the canula, but asphyxia being threatened, it was instantly abandoned. The larynx, therefore, was evidently still obstructed, although the infant had ejected by the canula nothing more than pinkish muco-puriform sputa, such as are seen in the bronchitis of measles. On the following days, we renewed our endeavours to remove the canula; but each time we took it out, it was necessary to replace it with the utmost possible haste, as no air traversed the larynx.

Ten days after the operation, the infant still had a favourable appearance, and continued to take the breast: she was in good spirits, her flesh had become firm, and she was not getting thinner:

but the canula had been kept constantly in the trachea. The lips of the wound were in a very good state. On the eleventh day after the operation, I was told that the patient had been very restless during the night. The pulse was quick, and the skin burning. The infant frequently took the breast, but immediately gave up her hold of it. Fine mucous râles were heard over the entire thoracic region. The fever, restlessness, and physical signs of general bronchitis caused me to form an unfavourable prognosis: very probably, small masses of pulmonary hepatisation already existed. An attempt was made to remove the canula, but it was fruitless, as the air did not traverse the trachea in sufficient quantity.

On the fourteenth day, the little patient, at the morning visit, lay motionless on the bed, and presented the signs of peripneumonic asphyxia. The pupils were very much dilated: sputa no longer came up through the canula: the pulse was too rapid to be counted: death, preceded by some convulsive movements, then closed the scene.

The autopsy showed that no pseudo-membranous deposit existed on any part of the respiratory passages; but the glosso-epiglottidean folds were red, and the aryteno-epiglottidean folds were œdematous. The opening of the glottis seemed to be almost entirely closed by the swollen, injected mucous membrane. The mucous membrane of the larynx, which was also injected, was the seat of an inflammatory vascularity. Similar indications of inflammation were seen on the mucous membrane of the trachea and bronchi. Several pulmonary lobules, particularly on the left side, were inflamed and purulent: when cut, they yielded, on slight pressure, small drops of pus: at the inflamed points, the lung had the appearance of a sponge filled with purulent matter. There was likewise empyema on the left side, which perhaps had had its starting-point in the nuclei of purulent hepatisation observed on the surface of the lung.

In this case, the pneumonia and pleurisy supervened during the last few days of life, the morbid changes advancing rapidly to the formation of pus.

Without any of the complications of an eruptive fever or pulmonary inflammation, false croup sometimes causes death; although generally, I may say very nearly always, it is not a dangerous affection. Fatal cases are quite exceptional; but you must recollect that they sometimes occur. Here is a case in point.

In 1834, I was summoned in haste to see a pupil at the College

of Juilly, who, I was told, was dying. The lad was thirteen years of age. On the evening before his attack, he was quite well. In the morning, on awaking, he was suddenly seized with a frightful attack of dyspnœa: he got up, however, and ran to the room of the prefect of the studies. Respiration was exceedingly embarrassed: there was a hoarse croupy cough: the voice was harsh and small: inspiration produced a noisy whistling. The medical attendant of the college, who was immediately sent for, was with good cause alarmed at the state of the patient, and at once sent off one of the masters to fetch me. I started forthwith: and in four hours reached the patient, who had just expired. The circumstances seemed to me to be too extraordinary to allow any means to be neglected to discover the cause of the sudden catastrophe. I removed with the greatest care the larynx and trachea and took them to my colleague's house, where we examined them. We found that it was only a case of false croup. There was a good deal of swelling of the vocal cords, redness of the laryngeal mucous membrane, and a little swelling of the aryteno-epiglottidean folds: on one of the vocal cords, there was a slight membranous concretion, possessing, however, none of the characters of diphtheritic false membrane, and depending upon a very intense inflammatory sore throat.

Patients, therefore, may die from stridulous laryngitis; but let me again repeat that such cases are exceedingly rare. During my long practice, I can only remember to have met with three fatal cases. Still, Gentlemen, I say, be reserved in your prognosis, notwithstanding the extreme benignity of the disease: in particular, be reserved, not so much because in a few very exceptional cases, the issue has been fatal, but because false croup may be the forerunner of catarrhal pneumonia, a disease which seldom spares those it seizes. I may say that I have had some experience in croupy affections, and though I am perfectly aware of the immense odds which the patient has in his favour, I cannot exclude from my mind a certain amount of dread that I may by-and-by have to do with that terrible disease of which false croup is only the first symptom.

As I began by stating, the differential diagnosis of true and false croup is a logical deduction from the facts which I have laid before you. Still, it sometimes happens, that to form a diagnosis is embarrassing: this is the case when stridulous laryngitis occurs in persons affected with common membranous sore-throat, and when diphtheria begins by simultaneously seizing larynx and pharynx.

You well know, that in the majority of cases, the membranous affection commences in the pharynx, and is thence propagated to the larynx: sometimes, however, though not frequently, the attack all at once begins with croup. When this occurs, it is very difficult to determine whether there is pseudo-membranous or simple laryngitis: but the progress of the symptoms may furnish presumptive evidence of the nature of the disease we have to deal with. Notwithstanding what has been alleged to the contrary, even in cases of sudden croup, the characteristic symptoms of the membranous affection are in general slowly developed: observe, I say, *generally* and not *always*: on the contrary, the characteristic symptoms of false croup quickly declare themselves. False croup sets in abruptly, and from the very first, the symptoms are alarming: but they decrease in severity. In true croup, the invasion is less abrupt, but the symptoms gradually go on increasing in severity. Suppose, for example, two children, one of whom has had a hoarse voice for two or three days, and a suspicious cough for forty-eight hours—the other, a sudden attack during the previous night of difficult breathing, accompanied by whistling inspiration, and a ringing croupy cough: of the two, the former is the most seriously ill; for he has true, while the latter has false croup. Diphtheritic inflammation, in fact, takes a certain time to evolve: two or three days elapse before it attains its maximum intensity. At first, the irritation which it causes in the parts about to be covered with false membrane is very slight, and only excites some fits of coughing: the discomfort in breathing, which in the first instance is produced by the swelling of the vocal cords, only shows itself by a moderate degree of oppression. It is not when the inflammation of the mucous membrane of the larynx is greatest, but when the thick diphtheritic membranous deposit interposes a physical obstacle to the passage of air, that there is most difficulty in breathing. Acute simple inflammation of the larynx proceeds otherwise: it almost at once produces swelling of the mucous membrane: in from half an hour to an hour, or in two hours at the most, this swelling is at its maximum, and the consequent sudden stricture of the opening of the glottis causes the suffocative seizures which characterise false croup.

It is a remarkable fact that the suffocative seizures occur during the night, and very seldom in the day time. To put it still more clearly, they take the patient by surprise when he is asleep, and not when he is awake.

Stridulous laryngitis not only differs from true croup in mode of invasion and progress of symptoms, but also, and even more, in the character of the cough called *croupy*, the semeiological value of which we have still to examine. On this subject, Gentlemen, let me give you the result of my long experience: it is not infallible—it is very far from being so—and so frequently does it deceive me, that I warn you not to be astonished should you be sometimes similarly misled: nevertheless, my experience has taught me things which it may be useful for you to know.

In very young children, however slightly the mucous membrane of the larynx is inflamed—and consequently swollen—the cough has a hoarse character, inspiration is whistling, and the voice is greatly altered. This is sometimes observed in simple catarrhal affections. The vocal cords are exceedingly sensitive to the mucus which falls on them, and even the air which traverses the glottis: in a word, the mucous membrane of the larynx, naturally of an irritable, excitable character, is in a state of exceedingly increased irritability. There will be incessant cough, and in the space of one minute, the patient will have from fifteen to twenty paroxysms. The cough, therefore, which has received the name of *croupy*, is in very young children, the consequence of acute inflammation of the mucous membrane of the larynx: or, to be more precise, it is the expression of the existence of simple acute inflammation. Diphtheritic inflammation, however, is not simple and acute: at first, it is very slight, and very much more superficial than simple laryngitis. If you will allow me to use the comparison, it is like the trifling superficial inflammation which accompanies malignant pustule, as compared with the severe inflammation which accompanies a common boil. Simple laryngitis makes a great *fracas*, but diphtheritic inflammation insidiously installs itself: the irritation which the latter causes in the parts which it invades, at first produces scarcely any fits of coughing, as I have just been remarking. To these slight symptoms a cough soon succeeds, which by its hoarseness and frequency recalls to one's recollection, those which I have just been pointing out under the name of *angina stridulosa*: at a later period, a pseudo-membranous exudation is found to have covered the vocal cords, the mucous membrane loses its sensibility, protected as it is by a sort of coat of mail from the action of air and mucus, by which otherwise its irritability might be excited. So much is this the case—and observers have been struck by the phenomenon—that in

confirmed croup [*croup confirmé*] there is little cough, and sometimes none at all: the cough is at least as often silenced as the voice.

This difference between the cough of stridulous laryngitis and of pseudo-membranous laryngitis is chiefly dependent upon entirely mechanical causes. If there be any notable structural alteration in the vocal cords, or if they be covered with a substance which cannot vibrate, there will be no vibration of the air as it passes over them. This can be shown by a very simple experiment. The larynx may be regarded as a wind instrument of the nature of a flute, or as an instrument having a tubal mouth-piece with expanded lips. Now, if we place a piece of moist parchment upon the lips of the mouth-piece, or on the openings in the flute, it will be impossible to obtain any vibration of air by blowing. When the cough remains hoarse and loud—*croupy* as it is called—in false croup, it is because the vocal cords are only swollen; and when it is muffled or extinct in true croup, it is because the larynx is covered with false membrane, producing an influence on the vocal cords similar to that produced on the metallic lips of the clarionet and bassoon.

Finally then—and that is the point I have been making for—*croupy cough is not an indication of croup*. Still, we can understand how the commencement of pseudo-membranous laryngitis may be invested with all the characters of false croup, and we can likewise perceive how it is, that when false membrane which covered the larynx has been expelled, the croupy cough should again be heard. Under such circumstances, however, it is soon again enfeebled, and finally, there is no sound: whilst this change is progressing, the severity of the suffocative symptoms increases. In stridulous laryngitis, on the contrary, as the cough loses its croupy character, the difficulty of breathing diminishes.

Cases are recorded in which stridulous laryngitis was accompanied by a feeble cough resembling the cough of true croup; but these very exceptional cases do not diminish the value of the differential diagnostic characters to which I now call your attention.

The remarks I have made on croupy cough are also applicable to changes in the voice. In croup, the voice is first of all very much altered in tone: it then becomes very much weaker, not only during the paroxysms, but also in the intervals between them. In false croup, if it become feebler during the paroxysms, the feebleness is never to such a degree as in true croup; and during the intervals it

regains its strength to a certain extent, though remaining hoarse and broken.

When stridulous laryngitis is coincident with common membranous sore-throat, formed by thick confluent patches of membranous deposit, however well-marked the laryngeal symptoms may be, hesitation is allowable: it is only by the subsequent progress of the case, that you can clearly establish your diagnosis. You must know, therefore, to wait before forming your opinion; but whilst you are waiting, treat the pharyngeal affection exactly as if it were diphtheritic.

An experienced practitioner will not mistake stridulous laryngitis for spasm of the glottis or thymic asthma; but as some authors have fallen into confusion on the subject, I shall rapidly point out the signs by which the differential diagnosis of the two affections may be established. We have just seen that in stridulous laryngitis there are suffocative seizures, that the cough and voice are croupy, and that during the whole course of the malady, even in the intervals between the paroxysms, the patients retain a certain amount of hoarseness in the cry, in the voice, and in the cough.

In spasm of the glottis, there is first of all this difference, that the paroxysms are equally liable to occur during the day and the night: then, again, when there are suffocative seizures, they are not accompanied by even slight hoarseness of cry, voice, or cough. Let me add, that the paroxysms do not generally recur two, three, or four times in the space of a few minutes, as is the case in false croup. However many attacks there may be in the twenty-four hours, there is always a long interval between them; and as soon as they are over, the patients breathe easily, retaining apparently no recollection of what they have suffered.

The treatment of false croup need not detain us long; for, as I have already told you, the disease cures itself. I shall only recall to your recollection the treatment adopted by Graves, of which I have already spoken in my clinical lectures on measles, and which consists in passing along the skin, under the child's chin and in front of his neck, a sponge soaked in very hot water. This operation is repeated several times at intervals of ten or fifteen minutes: it causes a sort of determination to the skin, under the influence of which the oppression usually ceases in a remarkable manner, while the cough loses its hoarseness. This powerfully efficacious treatment has the advantage of being marvellously simple; and it is usually sufficient

without any other means, such as emetics, being employed in connection with it. But even when there is no false membrane in the larynx, the swelling of the mucous membrane may be so great as to place life in immediate jeopardy, and to impose the necessity of performing tracheotomy. In such a case as this, my excellent friend Dr. Adolphe Richard restored to its mother, a poor child dying from suffocation, the consequence of stridulous laryngitis.

LECTURE XXVI.

ŒDEMA OF THE LARYNX.

Œdema of the Larynx is Not in itself a Disease: it is a Complication of Diseases of the Larynx.—Improperly named Œdema of the Glottis.—Sometimes, but not often, Independent of Inflammation.—Predisposing Causes.—Exciting Causes.—Frequently supervenes in Chronic Laryngitis.—Common Termination of what is called Laryngeal Phthisis.—Treatment:—Topical Medication is Important.—Often necessary to resort to Tracheotomy.

Gentlemen:—Some of you, no doubt, recollect a young woman of twenty-one who was brought to the Hôtel-Dieu, on the 24th June, where she occupied bed No. 20 of our St. Bernard's ward. She left the hospital perfectly cured, on the 2nd July, after having had all the symptoms of œdema of the larynx. She was, on a former occasion, for a month in our wards under treatment for puerperal peritonitis. She had been six weeks out of hospital, when she was seized with violent sore throat, difficulty of deglutition, and a good deal of swelling of the tonsils. When I first saw the patient, the affection had existed ten days, and had already made rapid progress. It soon produced an amount of difficulty of breathing, which gradually increased till it became so serious as to bring on suffocative paroxysms. During inspiration, the dyspnœa was accompanied by laryngo-tracheal whistling, but the expiratory sound remained normal, and the voice retained its natural tone. I found the patient with a good deal of oppression: the countenance was pale, and presented that peculiar expression observed in persons threatened with asphyxia. The pulse was small and miserable; the submaxillary region was swollen and painful. On examining the throat, I saw that the mucous membrane of the pharynx had a bright red colour: on carrying the finger towards the laryngeal orifice, I found that there was œdematous swelling of the epiglottis and aryteno-epiglottidean

ligaments. I had no hesitation as to the diagnosis: it was a case of what has been called œdema of the glottis.

I ordered an injection, as soon as possible, into the back part of the throat, of water-spray strongly charged with tannin. Similar injections were made every hour by means of the spray-apparatus which you know, and which has been modified by Mathieu. Under the influence of this medication, the severity of the symptoms moderated. At my second visit, on the following day, a great improvement was evident. During the whole day, there had been only one suffocative seizure, and it was of a much less violent character than those of the previous evening. The breathing had become free, and was not accompanied by any abnormal sound. There was also a great diminution in the swelling of the epiglottis and aryteno-epiglottidean ligaments. Notwithstanding the amendment, I ordered the treatment to be continued. For three days, he had one suffocative seizure in the twenty-four hours; but during the intervals between the attacks, respiration was natural. Although, at this date, the cure may be considered to have been complete, and the general condition very satisfactory, the patient did not leave the hospital till four days later, up to which time convalescence had been thoroughly maintained.

To-day, a new case of œdema of the larynx has been presented to our notice. The patient, a woman of fifty-two years of age, occupies bed No. 25 of the same ward. In her, œdema of the glottis is a sequel of chronic laryngitis. The recurrence of the seizures, their severity, and the imminence of the danger, necessitated surgical interference; and tracheotomy, the only means of preventing death, was resorted to with complete success.

You have also recently had an opportunity of interrogating a patient who occupied bed No. 23 of St. Agnes' Ward. This man, aged fifty-eight, who came into hospital on account of a deep-seated swelling of the lateral region of the neck, had on the anterior part of that region, two centimeters above the sternal *fourchette*, a linear cicatrix, the origin of which is thus accounted for. In 1858, he was under treatment, in the wards of my lamented colleague Dr. Legroux, for chronic laryngitis of syphilitic character. During fifteen days, he had been subjected to specific treatment, when suddenly, during the night, after a chill, he experienced great difficulty of breathing: next morning, the existence of œdema of the larynx was ascertained; as asphyxia was imminent, tracheotomy was at once performed.

The danger was averted; and in three days after the operation, it was found practicable to remove the canula. The patient was soon in a condition to resume the specific treatment, by which, in a few weeks, he was cured of the syphilitic laryngitis.

Gentlemen, I must not omit to speak to you of a formidable affection which you have had several opportunities of seeing in my wards during the last few years. When lecturing upon dothinenteria, I have already called your attention to œdema of the glottis, in connection with two patients who suffered from it, and in whom you had the opportunity to see the gradual development of the symptoms. In two other cases, I showed you, on the dead body, the larynx of persons who had sunk under tubercular phthisis, and in one of whom tracheotomy had been rendered imperative from œdema of the glottis, as in the case of the woman occupying bed No. 25 of St. Bernard's Ward. Finally, when speaking to you of scarlatina, I mentioned œdema of the glottis as one of the complications liable to supervene in the decline of that pyrexia.

By œdema of the glottis is meant a serous, purulent, or sero-purulent infiltration of the submucous cellular tissue of the epiglottis, and aryteno-epiglottidean folds, generally extending to the interior of the larynx. So accurate is this description of the affection called "œdema of the glottis," that every author who has written on the subject states that the diagnosis has to be made by an exploration of the parts by the finger. Now, however deep you may pass the finger into the back part of the throat, you cannot by any possibility get the finger to reach beyond the epiglottis and the aryteno-epiglottidean ligaments. Œdema of the glottis is, therefore, an incorrect name to apply to this affection, because in the majority of cases, its seat is not the glottis but the margin of its superior orifice. Again, the swelling of the aryteno-epiglottidean folds is itself a cause of symptoms which are otherwise very much more serious than those to which it gives rise when it only occupies the vocal cords. If you reflect upon the anatomical arrangement of the aryteno-epiglottidean folds, you will understand that, when they swell in so remarkable a manner as to form large cushions, trembling at each inspiration, as the air enters the larynx, they become glued to one another, closing by a sort of valve the upper part of the air-passage, while the vocal cords, being formed of a more compact tissue, and therefore not easily infiltrated, do not swell out in the same proportion. Those who have witnessed Czermak's experiments

with the laryngoscope have had an opportunity of convincing themselves, that during forced inspiration, the vocal cords diverge in such a manner as to form a very large opening.[1]

Although the name "œdema of the glottis," [*œdème de la glotte*] was, and still is, in constant use, "largyngeal œdematous sore throat" [*angine laryngée œdémateuse*] is preferable. Besides possessing the advantage of not falsely describing the seat of the affection, the name last mentioned expresses the peculiar character, without in any way asserting its pathological nature, an objection which applies to the term "submucous laryngitis" [*laryngite sous-muqueuse*], which has also been given to it, and which conveys the idea of an inflammatory malady. Œdema of the aryteno-epiglottidean is nearly always the result of inflammation, but it cannot be denied that in some rare cases, inflammation has either had no part or only a very secondary part in causing the œdema.

Some of you may recollect my stating on a former occasion, that scarlatinous anasarca may invade deep-seated parts, causing effusions in the serous cavities—pleurisy and pericarditis—also, œdematous infiltration of the veil of the palate, uvula, and aryteno-epiglottidean folds. In the lecture to which I refer,[2] I related the history of a child seen by me in consultation with my colleague, Dr. Henry. This patient having been suddenly seized, during the course of an attack of scarlatina, with considerable anasarca, would have been lost from œdema of the aryteno-epiglottidean folds, had it not yielded to cauterizations with the nitrate of silver and insufflations of alum into the back of the throat. I also laid before you the history of another case communicated to me by my colleague M. Richet. The patient, a child, was similarly affected; and was rescued from impending death by tracheotomy. To these cases may be added others published by Baudelocque and Barrier.[3]

These examples are more than sufficient to prove that there exists a non-inflammatory form of œdema of the glottis. In these cases, the infiltration takes place into the cellular tissue of the aryteno-epiglottidean ligaments, from causes similar to those which produce effusions in other parts of the body, without there having been preceding inflammation.

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[1] Czermak:—Du Laryngoscope. 8vo, Paris: 1860.

[2] See p. 186 of the Second Volume of this translation.

[3] Baudelocque:—Gazette des Hôpitaux, 1834.
Barrier:—Traité Pratique des Maladies de l'Enfance. T. 1er, p. 456.

I am aware that as an objection to illustrations derived from scarlatinous patients, it may be said, that in scarlatina there is always a pharyngeal inflammation, and that this inflammation being the cause of the œdematous congestion of the aryteno-epiglottidean ligaments, the œdema is consequently inflammatory in its nature: but to argue thus, would be to exaggerate the bearing of the facts. Might it not be said with equal justice, that the infiltration of the subcutaneous cellular tissue is produced under the influence of the inflammation of which the skin has been the seat during the eruptive period? Scarlatinous anasarca, however, does not occur during the eruptive period, but in the decline of the fever: moreover, it is by no means those who have had the eruption in the most violent manner, who are most frequently the subjects of anasarca; and again, the anasarca supervenes in patients who have not had the exanthematous eruption at all. In respect of œdema of the glottis, it is possible that the sore throat by which it has been preceded, favours its production, but if so, the pharyngeal inflammation is only the immediately exciting cause, the predisposing being here the principal cause.

It is reasonable to believe (although I cannot adduce examples in support of the view), that this non-inflammatory œdema of the larynx may take place in connection with every disease during the course of which we see anasarca supervene—in albuminuria for instance; but apart from these cases, idiopathic œdema of the glottis is far from occurring so frequently as some authors have alleged: and I repeat, that you will almost constantly see œdema of the larynx depending on inflammation, a fact which Bayle established, and was the first to describe.

Œdema of the larynx may be either primary, or consecutive: it is primary, when it is the result of an inflammatory action advancing towards the larynx or pharynx, and simultaneously to the aryteno-epiglottidean ligaments; and it is consecutive, when it depends upon an organic alteration of the larynx. In the first case, the inflammation is propagated to the seat of lesion, the aryteno-epiglottidean ligaments: in the second case, the serous infiltration is due to engorgement of the vessels connected with the diseased parts: but in neither, can the œdema of the larynx, as it has for its starting-point an ulcerated and consequently an inflamed tissue, be regarded as independent of inflammation.

What then are the different circumstances in which this œdema

supervenes? Before answering this question, let me say a word upon the conditions which favour the production of œdema of the larynx.

These conditions exist in the texture itself of the affected parts. You know, Gentlemen, that an inflammation of the skin, a common boil for example, causes swelling of the surrounding parts, which, within certain limits, retains the mark of the finger when pressure has been made upon them with it. Œdematous swelling, resulting from an afflux and effusion of fluids into the cellular tissue, will proportionately have the more tendency to be produced, the less the degree in which the tissue is compact. We therefore see it in the most marked degree in the eyelids and prepuce, when there is an inflammatory afflux to these parts; the presence of variolous pustules, for example, upon the eyelids, will determine a great amount of swelling in these membranous curtains, and, in the same way, a variolous pustule on the prepuce may occasion swelling sufficient to impede the passage of urine. Well then, the uvula, epiglottis, and aryteno-epiglottidean ligaments present identical conditions of structure, and as these organs are composed of a still looser cellular tissue, you can understand how it is that they have a tendency to become œdematous, not only under the influence of a direct attack of inflammation, but likewise from inflammation of neighbouring parts causing a stasis and consequent effusion of fluids.

Let us now review the different circumstances in which œdema of the larynx may supervene.

One morning fifteen years ago, when the physicians of the Necker Hospital were arriving for their visit, my honourable colleague Bricheteau and I were together in the vestry, when we were summoned in haste to a person just brought in, who was dying in frightful paroxysms of suffocation. He was a vigorous man of thirty-five or forty years of age, who had been picked up on the Boulevard des Invalides. Horrible anxiety was depicted in his countenance: his respiration was embarrassed to the very last degree; and during inspiration, he emitted a whistling sound from the larynx, while expiration was a little less difficult than inspiration. I at once introduced my finger deep down into the throat, and detected great tumefaction of the epiglottis and aryteno-epiglottidean ligaments. Interrogating the patient, who—though speaking with great difficulty, gave a good account of his state—we learned that on the previous evening, having drunk too freely at a wine shop,

he had been turned out into the street, where he fell asleep. The night was cold; and towards morning, he awoke with a violent sore throat, which was almost immediately accompanied by great difficulty in breathing: in an hour or two, it had attained the point at which we saw it. The pharynx was of a bright red colour, and the veil of the palate was much swollen: the enlarged uvula, more than three centimeters in length, trailing on the tongue, was infiltrated with serosity, and looked like a large yellow grape. This œdematous condition of the uvula led us to conclude that the epiglottis and aryteno-epiglottidean ligaments were in a somewhat similar state, and in fact showed us the nature of the case. We saw that we had to do with œdema of the larynx. Under the influence of a chill, the man had contracted a catarrhal sore-throat, a violent inflammation, which invading the whole of the throat, and extending to the entrance of the larynx, had attacked the epiglottis and aryteno-epiglottidean ligaments, in the same manner in which it had laid hold of the uvula and veil of the palate. Tracheotomy was performed; and in a few days the patient was cured.

In the young woman of bed No. 20, St. Bernard's Ward, of whom I spoke at the beginning of this lecture, the laryngeal affection which presented characters almost more alarming than those seen in the patient of the Necker Hospital, was also dependent on a catarrhal inflammation of the pharynx.

Thus, Gentlemen, you see that *catarrhal pharyngitis* may be one of the causes of œdema of the larynx. With that cause may be grouped *erysipelas of the pharynx;* whether the erysipelas be originally developed in that region, or in the face, it will extend to the pharynx. You will find two cases illustrating this point in the thesis of Dr. Laillier, to whom they were communicated by Dr. Gubler.[1]

Speaking in more general terms, I may say, that any inflammatory affection of the pharynx or back part of the mouth, irrespective of its particular nature and seat, may originate the affection now under consideration. A simple inflammatory sore throat, inflammation at the root of the tongue, or inflammation excited by the presence of a cancerous tumour of that organ will sometimes lead to œdema of the glottis, when the inflammatory afflux extends to the epiglottis and aryteno-epiglottidean ligaments.

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[1] Laillier:—Sur l'Œdème de la Glotte. [Thèse]: Paris, 1848.

The circumstances, however, in which œdema of the larynx is the consequence of inflammation descending from the parts above, or directly attacking the aryteno-epiglottidean ligaments are much less common than those in which it is the direct result of acute or chronic inflammation of the larynx itself.

We can understand the facility with which the fluxionary movement which accompanies acute inflammation of the larynx may extend to the ligaments of the epiglottis and even to the epiglottis, and determine in the cellular tissue which enters into their composition, a more or less considerable accumulation of serosity. This is chiefly observed in laryngismus stridulus, a form of laryngitis which is rare in adults and frequent in children. In that disease, it is not uncommon to see œdema of the mucous membranes not only originate in the larynx itself, but extend likewise to the aryteno-epiglottidean ligaments: the paroxysms of false croup also present the characteristic symptom of œdema of the larynx—whistling inspiration, and inspiration more laboured than expiration.

In describing to you the history of small-pox, I noticed the laryngeal complications met with in the eruptive stage of that disease: I mentioned three patients who were carried off by frightful suffocative paroxysms, and I stated that at the autopsy of one of them, appearances of inflammation were found in the larynx, and variolous pustules below the glottis. I am not aware of any cases having been recorded of œdema of the glottis dependent on small-pox; but looking to the cases to which I have now referred, one can quite well conceive that the affection may supervene in the course of small-pox, in consequence of pustules being developed upon, and in the neighbourhood of the aryteno-epiglottidean ligaments.

But the most frequent causes of œdema of the larynx, are the more deeply-seated affections of that organ. I refer to *laryngeal ulceration*, acute or chronic, embracing several species, which have been long known under the generic term *laryngeal phthisis*—to *non-specific* laryngeal ulceration—and to *syphilitic, cancerous*, and *tubercular* ulceration of the larynx.

Exclusive of the cases in which non-specific or what may be called idiopathic ulceration of the larynx supervenes after severe fevers (as in the two patients of whose cases I spoke when lecturing on dothinenteria), this species is rare. Generally, ulceration of the larynx is of one of the other species now enumerated, of which the

tubercular is the most common, and to which alone the name—the objectionable name—of *laryngeal phthisis* is at all applicable. Were this term to be rigorously and literally interpreted, we should say that it signified a chronic disease of the larynx capable in itself of giving rise to consumption. But it results from the anatomy of the parts, that patients sink most frequently under the œdematous affection before the disease has reached the last stage of marasmus.

Nevertheless, there are cases—very exceptional cases—in which death may be the consequence of *consumption*. At first it seems difficult to understand, how an inflammation of the larynx can of itself lead to consumption. We can conceive chronic inflammation, ulceration, or suppuration of the kidneys, intestines, bladder, or large extent of cellular tissue having the power of contaminating, day by day, the mass of the blood, exciting fever, and causing the patient to waste away; but we cannot so readily conceive such consequences being the result of similar conditions of the larynx, the morbid surfaces of which are so small in extent, the products of their suppuration so moderate in quantity, and their sympathetic relations so unimportant. But there is another point which here requires to be taken into consideration. The ulcerations burrow deeply, and extend into the larynx: the epiglottis and the arytenoepiglottidean ligaments participate in the inflammation: for a long time these parts are turgid, but not to a degree sufficient to cause complete obstruction to the passage of air: they have acquired excessive irritability: the larynx, of which the muscles and nerves have become pathologically modified, can no longer act in a normal manner. The patient breathes with difficulty: he is prevented from having a minute's sleep, and coughs incessantly from the irritating action of the air on the affected parts: cough is likewise brought on in fits, by the contact of alimentary substances and drinks, which, from difficulty of deglutition, are constantly getting entangled in the air-passages, and so exciting fears of suffocation. Under these conditions, the wretched sufferer refuses to take food, except when forced to do so by the imperious demands of hunger. His nutriment, therefore, is insufficient, and he falls into a state of emaciation which leads him to his grave.

Laryngeal phthisis, I repeat, is quite an exceptional termination of laryngeal ulceration. The most frequent cause of death is œdema of the larynx, resulting from previous disease of the larynx.

Whether the inflammation of the larynx be simple or syphilitic,

tubercular or cancerous, œdema may supervene, when there are more or less extensive and more or less numerous ulcerations.

Thus it happens, Gentlemen, that we frequently meet with individuals, who, having to a certain extent lost their voice from syphilitic disease, continue to speak with more and more difficulty, and increasingly to suffer from difficult breathing. The dyspnœa, which at first existed only when they exerted themselves in some unusual manner, such as walking more quickly than usual, or ascending a stair, at last becomes permanent, being present even when they remain in complete repose. Inspiration, which is more embarrassed than expiration, is accompanied by a characteristic laryngeal whistling; and the symptoms go on increasing in severity from day to day, till real suffocative attacks supervene. By introducing the finger behind the tongue, the condition of the epiglottis and aryteno-epiglottidean ligaments can be ascertained: by this proceeding it will be found, that they are swollen, and in a state of œdematous puffiness. This œdema of the glottis is dependent upon an inflammation of the larynx, characterised by primary or secondary syphilitic ulcerations, whether the disease has begun in the larynx or pharynx.

The same phenomena are observed in persons affected with tubercular laryngitis, of which the most common lesions are *erosions*, involving only the mucous chorion, or *ulcerations*, presenting great variety in number, form, extent, and depth. The number of the ulcerations is generally in an inverse ratio to their size, although it is by no means unusual to find a single, very small ulcer on the margin of the vocal cords, or at the bottom of one of the ventricles. Ulcers may invade the entire larynx, vocal cords, aryteno-epiglottidean ligaments, and mucous membrane of the epiglottis: of the latter, Dr. Belloc and I, in our treatise on laryngeal phthisis, have given an account of a remarkable example, and illustrated it by a drawing. With regard to form, the ulcerations are sometimes rounded and are sometimes irregularly circumscribed: their edges are at times jagged, and at other times flattened: their depth also is variable. In the majority of cases, the ulceration evidently begins in the mucous membrane, but in others, we meet with sub-mucous abscess, when it is clear that the ulceration is produced in the same way in which some cutaneous fistulæ are formed. Under the influence of an unobserved cause, or in consequence of irritation excited by exposure to cold, a more acute inflammation supervenes around the ulcerations; the fluxionary movement is propagated to the aryteno-

epiglottidean ligaments, and serous infiltration into their cellular tissue takes place, the symptoms of œdema of the larynx being thereby produced.

When the laryngitis—whatever may have been its cause—has led to necrosis or caries of the cartilages, œdema of the glottis is inevitable: under such circumstances, it is, so to speak, a necessity.

These structural changes of the cartilages of the larynx occur in simple ulcerous laryngitis, as, for example, in those which follow aggravated fevers. In relation to this point, I ask you to recall to your recollection cases which we observed together, and on which I dwelt too long when reviewing the sequelæ of dothinenteria, for it to be necessary now to repeat what I then said. These changes are met with in syphilitic ulcerous laryngitis. They are most common, however, in tubercular ulcerous laryngitis.

The ulcerative process, burrowing deeply, reaches the cartilages, which it denudes; then, according to the greater or less rapidity of the ulcerative inflammation, there is found, either necrosis without previous ulceration of the cartilages, or caries of the cartilages; or in other cases, there is ossification along with necrosis. Necrosis without ossification is observed in acute ulcerous laryngitis following aggravated fevers: caries, which I have never seen coincident with tubercular laryngitis, is almost invariably observed in very young subjects; while in persons of more advanced years, when the laryngitis has been of long standing it is necrosis that we find, and it is always accompanied by ossification, the latter, in fact, has even preceded the necrosis, the ulceration which is the cause of the necrosis having commenced by determining inflammation of the perichondrium, and a subsequent exudation of osseous matter into the subjacent cartilage: then, ulceration reaching the ossified cartilage, it becomes the more readily necrosed, that ossification has deprived it of a great part of its vitality. In old people, in whom ossification of these cartilages has pretty generally taken place, simple chronic laryngitis, irrespective of any special diathesis, leads to the structural changes of the larynx of which I have been speaking; consecutively also to œdema of the glottis, as occurred in the patient occupying bed No. 25, St. Bernard's ward.

Gentlemen, you can understand that when once necrosis of the cartilages has begun, œdema of the larynx inevitably follows. Here, as in necrosis of the bones, where the sequestra must be extruded, as also in all tissues, in the cellular tissue, for instance, where the dead

portions must be separated from the living—the rapidity with which the separation and elimination take place is proportionate to the vitality of the tissues.

In respect of bones, what takes place? The irritation caused by the sequestrum induces suppurative inflammation; and if the necrosis be subcutaneous, the pus, sooner or later, makes its way to the surface. Sometimes, the opening becomes cicatrised, but if this occur, it soon reopens, unless other openings, form in the neighbourhood to afford an exit to the pus which is being constantly secreted: finally, a fistula is formed, which continues to exist till the whole of the dead portions of bone have been eliminated. The inflammation extends to the soft parts, which swell, and become the seat of œdematous engorgement.

In necrosis of the cartilages of the larynx, the course of events is similar. As soon as there is necrosis, whether of the cricoid cartilage, which is most common, or of the thyroid cartilage, which is less usual, an absolute necessity exists that the necrosed part be eliminated. During the whole of the period of elimination, suppuration is going on: abscesses form under the laryngeal mucous membrane, which they detach, and from the inflammatory afflux extending at the same time to the neighbouring cellular tissue, that becomes the seat of more or less pasty induration.

To pursue still further our comparative study of the manner in which bone and cartilage is affected, let us suppose an individual with necrosis of the tibia suddenly taking erysipelas of the leg, under the influence of one of those epidemics so common in our hospitals. This attack will have as its starting point the existing fistula; and the erysipelas, acquiring great intensity, will occasion an engorgement extending to a greater or less distance around the primarily affected parts.

Suppose, again, that an individual suffering from ulcerous laryngitis and necrosis of the cartilages, take acute inflammation of the larynx, from exposure to cold, undue exertion of the voice, or other cause, that inflammation, being greatly aggravated by that already existing, will extend to distant parts, will reach not only the vocal cords, but likewise the aryteno-epiglottidean ligaments; and the patient will have all the symptoms of œdema of the larynx.

I ought also to notice a frequent cause of œdema of the glottis in very young children in England, North America, and Russia, where tea is an ordinary beverage. In every family there is almost con-

stantly a kettle on the fire : and the children going to drink from the beak of the kettle or tea-pot when the boiling water is about to be poured out, terrible burns of the mouth and throat are the result. No doubt the child immediately rejects the water which he has taken into his mouth ; but it has had time to come in contact with the epiglottis, the aryteno-epiglottidean ligaments, the veil of the palate, and the interior of the mouth. In general, for some hours after the occurrence of accidents of this description, they do not seem to be at all serious; but ere long, respiration becomes distressed, and all the phenomena of œdema of the glottis make their appearance.

Mr. Jameson, surgeon to one of the Dublin Hospitals, has published a very interesting paper on this subject. He shows the necessity of resorting to tracheotomy as soon as the suffocative attacks occur ; and he details several cases in which a cure was obtained by that proceeding. The canula ought to be removed as soon as the local effects of the burn have disappeared.[1]

You know, Gentlemen, the *symptoms* of œdema of the glottis. In a few rare cases, the disease declares itself suddenly, as occurred in my patient of the Necker Hospital. More frequently, according as it is dependent on an acute or chronic inflammation, the phenomena by which it is characterised have been preceded by the symptoms which belong to these diseases, that is to say, by the symptoms of pharyngitis, tonsillitis, acute laryngitis, or chronic laryngitis.

In the latter, the most frequent case, *changes in the voice* will have existed for some time : the individual will have had for some time roughness of voice which will at last pass into aphonia : his hoarse, dry cough will become less and less heard, till it ultimately becomes inaudible. At this stage, the disease of the larynx having made progress, *respiration* will have become more *painful*. At first, the oppression is greatest during inspiration, which is accomplished with great effort, and is accompanied by a guttural snoring noise, which is sometimes very loud : at first, this noise is only heard during sleep. Expiration, hitherto easy, now, in its turn, becomes difficult. The malady advances, and the dyspnœa increases. The dyspnœa, which in the beginning of the attack was most severe at night, is now great both by day and by night ; but during the night it is so urgent, that the patients are obliged at last to retain constantly the sitting posture. *Orthopnœa* becomes incessant ; and it has exacerbations

[1] Jameson :—Dublin Quarterly Journal for February, 1848.

which are suffocative seizures, and constitute the symptom characteristic of œdematous sore throat.

These suffocative seizures have a very frightful aspect. The patient—with livid face, open mouth, distended nostrils, moist and protruding eye, the skin streaming with sweat—rises abruptly, and walks about the room, from time to time holding the articles of furniture, the jams of the mantelpiece, or the ratteens of the casements, seeking everywhere something to rest on that he may breathe more easily: sometimes, he will hold his head low and look down; but more frequently, he will, with stretched neck, turn his head backwards: at last, he will sit down exhausted, but he will soon rise again and repeat the same postures. You see him in extreme excitement, throwing off the garments which cover his head, neck, and chest, opening the windows in a sort of frenzy, that he may inhale the fresh air from without, and grasping his neck, as if for the purpose of wrenching from it some foreign body by which he was being strangled.

Persons sometimes die in the first paroxysm of œdematous sore throat; but in general, the symptoms abate, and the suffocative attack ceases: the breathing, however, continues embarrassed, particularly during inspiration: the voice is scarcely audible: the state of excitement is succeeded by collapse.

When we proceed to examine into the state of the affected parts, we find that our means of investigation are unfortunately very insufficient. If in certain cases, *inspection of the back part of the throat* is of some use; if the existence of a catarrhal or inflammatory sore throat lead us to believe that the œdema of the glottis is dependent upon pharyngitis, this inspection too often fails to aid our diagnosis when the sore throat which preceded the laryngeal affection has entirely disappeared, or when the affection of the aryteno-epiglottidean folds is dependent upon disease of the larynx. In the latter case—which is the most usual—auscultation does not afford information as to the state of the parts nearly so good as that obtained by observing the manner in which respiration is performed, and attentively studying the modifications of the voice. It is only *the touch* which can give us some positive indications; but to whatever degree of perfection this mode of exploration is carried, it does not do more than enable us to recognise the œdematous swelling of the epiglottis and aryteno-epiglottidean ligaments. Exploration by the finger must be practised in a very careful manner. You recollect

that whilst I was examining the throat of a woman with my finger in the most guarded possible way, I induced a suffocative seizure which very nearly proved fatal. To ascertain the existence of lesions is unquestionably of great importance in the diagnosis of œdema of the larynx, but it throws no light upon the nature of the affection whence the œdema arises. Examination of the larynx by a suitable speculum was felt to be a likely means of attaining this end. Long prior to 1837, when Dr. Belloc and I published our treatise on laryngeal phthisis, this idea had engaged the attention of practitioners; and at the date of our publication we were occupied with the construction of a *speculum laryngis*. At that time likewise M. Selligue, an ingenious mechanician, who was also a sufferer from laryngeal phthisis, made for his physician, an apparatus consisting of two tubes, one for throwing light on the glottis, and the other for affording a view of the image of the glottis, as reflected in a mirror placed at the guttural extremity of the instrument. There were, however, serious defects in this instrument; and the difficulties in applying it were so great, that I long since ceased to use it. Laryngoscopy has been carefully studied in England and Germany; and you can read in the *Archives Générales de Médecine* for February 1860 an account by my friend Dr. Lasègue of the results arrived at by our colleagues on the other side of the Channel and beyond the Rhine. When laryngoscopy shall have attained a greater degree of perfection, it will no doubt render service not only in the diagnosis but also in the treatment of laryngeal affections—particularly in the treatment of œdema of the glottis, for sight ought certainly to assist the hand in the application of the topical remedies which are of such essential importance in treating that affection.[1] I must not, however, exaggerate the practical utility of the laryngoscope in the class of cases we are now considering. The application of instruments is not well borne by the larynx, particularly when there is a liability to suffocative seizures; and you have observed that M. Czermak, notwithstanding the great experience which he had in the use of the laryngoscope, was only able to get a good view of the larynx in patients with very tolerant throats, and in whom respiration was not much embarrassed. When there is suffocation, the introduction of the laryngoscope increases the anxiety,

[1] See the work of CZERMAK:—"Du Laryngoscope et de son emploi en Physiologie et en Médecine." Paris, 1860.

and it is only by stealth, if I may use such an expression, that one can get a view of the vocal cords and upper part of the larynx.

I now propose to consider the symptoms and progress of œdema of the larynx.

I stated, Gentlemen, that patients are occasionally carried off in the first suffocative seizure; but that usually this attack passes off, leaving, however, embarrassed breathing. I also stated, that there is a variety in the early symptoms, according to the œdema of the larynx being dependent upon acute or chronic inflammation. In the former case, the attack comes on abruptly, and the symptoms are of a violent, irregular character, recurring several times within the twenty-four hours; in the second case, the seizures supervene at distant intervals, at intervals of from eight to fifteen days, or longer; but after a time, the duration of the intervals diminishes, so that in the course of the twenty-four hours, there are several attacks, those which occur during the night being the most violent.

When œdema of the larynx is a primary affection, or is connected with acute inflammation of the pharynx or larynx, its progress is more rapid, and the chances of a favourable termination are also greater, which arises from the affection being transient in its nature like the pathological state on which it depends. Spontaneously, therefore, or under the influence of appropriate treatment, resolution of this inflammation takes place, the cure being certain and complete. I am not speaking to you, Gentlemen, of very exceptional cases in which recovery is the result of another mechanism which you will find pointed out in some works—the opening of an abscess formed in the substance of the aryteno-epiglottidean ligaments.

When œdema of the larynx is connected with chronic inflammation of the larynx, or structural alteration of the cartilages, the progress of the symptoms is very different; and from what I have already said, you can understand that they then have a disposition to repeat themselves. There is in fact an inevitable necessity for the elimination of the necrosed parts: the eliminative process gives rise to suppuration, and to the formation of abscesses, which by raising up the mucous membrane, diminishes the calibre of the glottis, already narrowed by the thickening of the vocal cords caused by their inflammatory engorgement; and which engorgement, by extending to the aryteno-epiglottidean ligaments, causes them to become infiltrated.

If the pus make an exit for itself into the interior of the larynx,

or externally by the skin, (examples of which I have seen,) if the inflammatory action is at first very circumscribed, and quite passes away, the suffocative symptoms will decrease more or less completely, in proportion to the greater or less size of the opening in the abscess, the patient, however, retaining hoarseness of voice, some amount of aphonia, and difficulty of breathing, which latter depends on the vocal cords and laryngeal mucous membrane remaining in a thickened state. But this amendment is only temporary: the causes continue, and will, sooner or later, induce a repetition of the consequences. The organic lesion advancing and the œdematous infiltration becoming permanent, the symptoms recur with increasing intensity; and unless art interpose, the patient will probably be carried off in a suffocative paroxysm. Death also frequently occurs in the intervals between the attacks. The patients becoming weaker and weaker, more and more prostrated by each attack, fall into a state of drowsy listlessness, and sometimes expire in perfect possession of consciousness. In such cases, tracheotomy often gives no fresh life, the victims sinking, in the same manner as certain asphyxiated persons sink, after the causes of asphyxia have been removed.

Although the principal obstacle to respiration in œdema of the larynx is generally seated in the aryteno-epiglottidean folds, this inflammatory œdema may also become developed in the cellular tissue of the laryngeal mucous membrane itself: thus, in the cases in which there is none of that shrill whistling inspiration which particularly belongs to œdema of the aryteno-epiglottidean folds, it is probable that the chief obstacle exists in the situation of the vocal cords. Then, the patient, who generally has serious lesion of the cartilages of the larynx and particularly of the cricoid cartilage, has no whistling inspiration: there is puffiness of the mucous membrane and œdema of the submucous cellular tissue covering the affected cartilage: there is only very greatly embarrassed breathing, the inspired air traversing the larynx, and producing there a more or less harsh but not shrill sound, while the expiratory murmur is still perceptible: there exists in fact a variety of wheezing without any shrill whistling sound. You may have observed this state of matters in a patient occupying bed No. 3 in St. Agnes' ward. The man to whom I refer is sixty-three years of age: for a long period he had been the subject of chronic laryngitis. Examination by the laryngoscope enabled us to ascertain that there was no œdema of the aryteno-

epiglottidean folds, while we saw diffuse redness of the upper part of the larynx and vocal cords, and below them, a serious structural change. We were ignorant of the cause of the chronic laryngitis: no benefit had resulted from treatment, and the embarrassment in respiration was rapidly increasing. Suffocative seizures frequently occurred during the night, the extremities at the same time becoming cold. Fearing that the patient might die in one of these attacks, I requested M. Dumontpallier to perform tracheotomy. The operation is, as you know, difficult in old people. In them, the trachea is nearly always very deeply seated. The large veins must be avoided with care, lest hemorrhage complicate the proceedings. When the trachea has been properly isolated, the most difficult part of the operation remains to be encountered—the opening of the canal, which is generally ossified. The upper rings of the trachea ought to be included between the blades of the scissors, care being taken to avoid cutting the mucous membrane. Before penetrating the mucous membrane of the trachea, it is necessary to remove a portion of the rings, a precaution without which it is impossible to introduce the canula. In the aged, therefore, there is a particular time for opening the mucous membrane of the windpipe, and that time is the last stage of the operation. In our patient, tracheotomy was performed in accordance with these rules, and the canula was easily introduced. The patient at once breathed freely: and no untoward event occurred after the operation. I said to you at the time, that it would probably be right to leave the canula in the trachea, because, whether the affection was cured or not, such an amount of stricture would remain as to leave an insufficient passage for the entrance of the air required in hæmatosis. Well then, the man, after remaining six months in our wards, was received as an incurable into the Bicêtre. He could not remain more than a minute without the canula, and when it was being cleaned, it was necessary to keep the tracheal opening in a patent state by means of the dilator, for otherwise a risk of suffocation would be incurred. From time to time, small sequestra from the cricoid cartilage were eliminated.

I now come to the subject of *treatment*.

When the inflammation is simple and very acute—when, as in our patient of the Hospital Necker, it is connected with violent inflammation of the pharynx or larynx—when the febrile reaction is intense—antiphlogistic remedies are at once indicated. One or two

large general bleedings, and the abstraction of blood from the cervical region by leeches or cupping, will give great relief and moderate the severity of the symptoms. Recourse will afterwards be had to cauterization with nitrate of silver, to insufflation of alum or tannin into the back part of the throat, and as far down as the aryteno-epiglottidean ligaments. When it is practicable, scarification of these ligaments has been recommended. I have not had the courage to practise this operation; but Mr. Gordon Buck, surgeon to the hospital of New York, has published numerous cases of recovery from œdema of the larynx in which repeated scarifications of the epiglottis and aryteno-epiglottidean ligaments were performed. The instrument employed, a sort of blunt-pointed bistoury with a short curved blade, is carried to the back of the throat, the index finger being used as a conductor. Mr. Gordon Buck has, however, exaggerated both the utility and the facility of this operation. In reading over the cases reported by this honourable practitioner, it may be asked, whether the majority of the patients would not have recovered under simpler treatment. The cases, Gentlemen, were acute, non-symptomatic cases of œdema of the glottis, an affection, which as you know corresponds in the adult to false croup in the child, and which gets well of itself, notwithstanding the very alarming aspect of the symptoms.

Topical treatment is exceedingly useful. You saw me employ it successfully, and to the exclusion of other remedial means, in the case of the patient of bed No. 20 St. Bernard's ward: in that case, there was laryngeal œdematous sore throat dependent on catarrhal inflammation, unattended by much general febrile disturbance of the system. Let me call your attention to the manner in which I applied the treatment. It consisted in injecting (by means of a spray-apparatus) water-spray strongly charged with tannin. This excellent method of application, which is easy in adults, is in my opinion even more serviceable in chronic affections of the larynx, than in œdema of the glottis.

Whatever may be the nature of the disease, whatever may be the laryngeal lesion which has induced the œdema of the glottis, the first thing to do is to apply topical treatment. Under its influence, the local affection of the aryteno-epiglottidean folds may become modified to such a degree as to cause a cessation of the symptoms, and gain time sufficient for the inflammation which originated the œdematous infiltration to pass through its stages, and come to an

end. Recollect, Gentlemen, that a definitive cure cannot take place, unless the pharyngeal or laryngeal inflammation upon which the œdematous laryngeal sore throat depends is of the kind which spontaneously terminates in recovery — unless the inflammation be either simple in its nature, or dependent upon a diathesis the manifestations of which we can prevent.

Let me explain this point. We are justified in hoping for a radical and final cure, when œdema of the glottis supervenes in the course of a simple ulcerous or syphilitic laryngitis, because in these cases we possess the means of effectively combating the pathological condition of which the œdematous affection is the result: but we cannot entertain such hopes when the œdematous affection comes on in the course of a tuberculo-ulcerous, or a cancerous laryngitis, for then the disease of the larynx is developed under the influence of a diathesis beyond the resources of art. Supposing that we are fortunate enough to master the symptoms of œdema of the glottis, we must be prepared for their return.

These considerations are still more applicable to cases in which œdematous laryngeal sore throat depends upon serious lesions of the cartilages of the larynx. When lecturing on dothinenteria, I mentioned the case of a young woman who became affected with œdema of the glottis after an attack of typhoid fever, and was entirely relieved after getting rid of some small osseous sequestra. This is certainly the most fortunate termination which can be met with; but it occurs too seldom to be counted on. The process of eliminating the necrosed parts is accomplished too slowly—in the case to which I alluded, the laryngeal malady was of nine months' standing—to prevent fear of the patient being carried off by a suffocative attack. When the œdema of the larynx depends on formidable lesions of the laryngeal cartilages, it is necessary to wait to see whether there be a recurrence of the symptoms. Here, the employment of topical treatment is explicitly indicated, because it will, by gaining time, afford the slight chance which there is of the fortunate result occurring which I have just mentioned. Sooner or later, however, it will be necessary to resort to tracheotomy.

In conclusion, I shall now repeat what I said on a previous occasion when speaking on this subject. When we have to deal with patients affected with œdematous laryngeal sore throat, after we have tried the therapeutic means at our disposal, insufflation of tannin and alum, cauterization with nitrate of silver, and (when

practicable) scarification of the aryteno-epiglottidean folds, we must hold ourselves in readiness to perform tracheotomy. Earlier or later, recourse to the operation will be determined by the severity of the suffocative seizures, the rapidity with which they follow one another, and the urgency of the dyspnœa in the intervals between the paroxysms. Finally, increased debility of the patient demands earlier recourse to the operation.

LECTURE XXVII.

APHONIA:—CAUTERIZATION OF THE LARYNX.

Different Causes of Aphonia.—From Lesion, or without Lesion of the Larynx.—Nervous Aphonia.—Good Effects resulting from Cauterization, and sometimes even from the mere Introduction of the Laryngoscope.

Gentlemen:—A long period has now elapsed since I called the attention of practitioners to a mode of treatment which I had found wonderfully successful in certain cases of chronic aphonia. During the present year, you have had an opportunity of judging of its efficacy, having seen me apply it in the cases of several young women who remained in our wards for some days.

By the term *aphonia*, we mean a more or less complete loss of voice, the power of speaking remaining. The patient has not lost the power of uttering articulate sounds, as in dumbness, with which aphonia must not be confounded; the sound of the voice is only greatly enfeebled.

The causes of aphonia are numerous, and also various in character. Chronic aphonia is generally a symptom of disease of the larynx, and is most frequently dependent on ulcerous laryngitis, of which I incidentally spoke in my last lecture. You will often meet with it in persons who have formerly had venereal symptoms, and it almost always occurs in tubercular laryngitis.

According to one of my good pupils, Dr. Krishaber, whose researches have rendered him a very competent authority on the subject, syphilitic leads less frequently than tubercular laryngitis to complete aphonia, and the explanation of this is to be found in the nature of the lesions. In syphilitic laryngitis, the lesions generally occupy, in order of invasion:—1st, the epiglottis; 2nd, the superior thyro-arytenoid ligaments (or superior vocal cords); 3rd, the arytenoepiglottidean ligaments (the seat of suffusion, œdema, and even of suppuration); 4th, the mucous membrane of the vestibule of the

larynx; 5th, the mucous membrane of the trachea; and 6th, (quite as an exceptional occurrence) the inferior thyro-arytenoid ligaments (or inferior vocal cords.) You will immediately perceive, Gentlemen, that this last peculiarity explains the rarity of complete aphonia in syphilitic laryngitis. Dr. Krishaber adds, that when the aphonia is complete, it is almost never dependent on a lesion of the vocal cords properly so called, but is generally the result of the swollen, puffy superior vocal cords covering, and preventing vibration of, the inferior vocal cords.

With the aid of a strong light—sun-light, or electric-light—we can see the colour of the mucous membrane, and from that determine whether the lesion be due to syphilis or tuberculosis: if the mucous membrane have a dusky shade, the affection is syphilitic.

It is more difficult to recognise the specific character of the lesion by the form of the ulcerations, as in both lesions it is very similar. The syphilitic ulcerations are deeper, and more frequently attack the cartilages, while in phthisis, the fibro-cartilaginous tissue alone is implicated. Œdema of the larynx is more common in ulcerous laryngitis depending upon syphilis, than in laryngitis arising from the tubercular diathesis.

Tubercular laryngitis invades in succession :—1st, the mucous membrane of the superior thyro-arytenoid ligaments; 2nd, the epiglottis; 3rd, the inferior vocal cords; 4th, the arytenoepiglottidean ligaments; 5th, the vestibule; 6th, and exceptionally, the mucous membrane of the trachea.

Syphilitic laryngitis shows a tendency to the formation of polypiform vegetations, which are met with throughout the whole extent of the larynx and trachea. In tubercular laryngitis, on the contrary, there are no vegetations; but a peculiar appearance resembling a polypus is produced by puckering of the edges of the ulcerated mucous membrane.

Cases of purely *nervous aphonia*, it is important to remember, are sometimes met with in both diatheses. I shall have forthwith to speak of this class of cases.

Dr. Krishaber has stated, in an oral communication, that there is most destruction of parts in simple, that is to say, in non-diasthetic ulceration of the larynx, as, for example, in that which originates in chronic laryngitis, or occurs as the sequel of a severe attack of fever. When speaking to you of œdema of the larynx, I stated that it is in

simple chronic laryngitis, in the ulcerous laryngitis consecutive to dothinenteria that, nearly always, necrosis of the cartilages, and subsequent symptoms of the most formidable character, occur.

Lesion of the recurrent nerves, accidental deformity of the larynx, the compression of that organ by a cervical tumour, or abscess, or the existence in its interior of vegetations, fungous growths, and polypi, may occasion aphonia: but it is not unusual to meet with it when there is no serious anatomical lesion; and in such cases, the affection is not less obstinate, lasting as it sometimes does for years.

The method of treatment of which I wish to speak to you to-day, and which is not applicable to, or at least is of very little service in, aphonia dependent on a serious affection of the phonetic apparatus, is exceedingly useful when the aphonia is independent of serious material lesions, and still more beneficial in those cases in which no lesions at all can be observed.

There are two species of aphonia, distinguishable from one another by the manner in which the symptoms are developed. The one comes on gradually: from time to time, the voice is observed to be muffled, soon after which, it becomes increasingly hoarse. The tone of the voice is very deep when the patient rises in the morning, and in the evening, it is much more shrill. At this stage, it is only by making great efforts that clear sounds can be produced; subsequently, there are days when, after too much speaking, no effort avails, and the larynx absolutely refuses to produce any sounds. At first, this species of aphonia, which is intermittent, comes on chiefly in the evening: it afterwards becomes complete and continuous. It affects both sexes; but men are most subject to it. It is chiefly met with in persons who have to cry aloud, to sing or speak in a high pitch in the open air or in a large expanse. Consequently, singers, advocates, clergymen, naval officers, and itinerant hucksters are frequently subject to serious alterations in the tone of the voice, and at last to aphonia. This species of aphonia is often coincident with chronic follicular inflammation of the pharynx: the probability is that the inflammation extends from the pharynx to the mucous membrane of the larynx. Though in such cases, the lesion may be superficial, it has not less the power to alter very much the tone of the voice; and as it generally is a symptom of the existence of the herpetic diathesis, it is particularly obstinate. Nevertheless, arsenical fumigations, followed, at a later stage, by cauterizations of the upper part

of the larynx, are generally sufficient to bring about permanent recovery.

An engineer on the Spanish railways consulted me regarding an affection of this kind. He did not derive much benefit from the iodide of potassium; but his condition rapidly ameliorated from the use of arsenical cigarettes, made according to a formula which you know, and which is as follows :—

Take of Arseniate of Potash	.	1 gramme, [15 grains.]
,, Distilled Water	.	15 grammes, [15 fl. drachms.]

Evaporate this solution upon a sheet of white paper which does not contain any glue. Having dried the paper make it into twenty cigarettes.

Every morning, the patient ought slowly to inhale into the bronchi from eight to ten whiffs of the smoke of one of these cigarettes.

Along with that treatment, I applied, every second day, to the upper part of the larynx, a very small sponge, fixed to the end of a piece of bent whalebone, slightly soaked in a saturated solution of sulphate of copper. From time to time, I substitute tincture of iodine for the sulphate of copper. Eight days of this treatment produced a remarkable improvement in the state of the patient. By the end of the month, the voice was quite restored. I then directed the patient to have recourse to the cigarettes on eight continuous days, once a month, with a view to prevent a relapse.

There is another species of aphonia, which begins abruptly, and without having been preceded by any other laryngeal affection. It is caused by a great shock to the nervous system : it is liable to occur in very irritable subjects, and particularly in hysterical women, in consequence of some violent moral emotion, such as fear, anger, bad news, or great joy. All of you know the classical story of the woman who, from finding her husband in the very act of adultery, suddenly lost her voice.

Classical authors have hitherto taught that nervous aphonia is solely dependent on lesion of the recurrent nerves. Dr. Krishaber remarks that they seem all to have forgotten that the superior laryngeal nerves only supply the crico-thyroid muscles—the chief phonetic muscles. These muscles render the vocal cords tense by causing the thyroid to swing upon the cricoid cartilage. They

impart to the inferior vocal cords the oscillatory movements observed, by the aid of the laryngoscope, when the voice is normal.

Aphonia, therefore, may either be dependent on a lesion of the superior laryngeal nerve or of the inferior laryngeal nerve, or on a simultaneous lesion of both nerves.

When the superior laryngeal nerve only is affected, the aphonia is never complete: the patient can generally articulate the deep tones, and his voice is hoarse, but he cannot utter the higher notes. This statement is both supported and explained by the experiments of Charles Bernard. In fact, the section of the superior laryngeal nerves causes the voice to become hoarse, but does not produce its complete extinction: the hoarseness depends on paralysis of the crico-thyroid muscles, and an inadequate tension of the vocal cords. In patients thus affected, laryngoscopic examination demonstrates this fact in the most unquestionable manner. Thus it is, that although we can see the vocal cords approach and separate in a normal manner, the emission of sound is accomplished with difficulty. One is struck with the absence of oscillation of the vocal cords, which is indispensable to the production of normal voice, and which is dependent on the action of the crico-thyroid muscles.

When the inferior laryngeal nerve is affected the aphonia is complete; and the experiments of Ch. Bernard have shown that this is the case whether the affection be with or without lesion. By the aid of the laryngoscope, we can see that the vocal cords are motionless and far apart; if there be any movement, it is very slight, and connected with respiration.[1] The following is a case of nervous aphonia, due to lesion or functional disturbance of the superior laryngeal nerve.

A young woman, aged twenty-seven, a shoe-merchant, presented herself as an out-patient in the consultation-room, during May 1863. She complained of an alteration in her voice, which was very hoarse; and when she attempted to utter the higher sounds, she suddenly lost her voice altogether. Dr. Krishaber examined her with a laryngoscope in the presence of myself and my clinical class. He found that the appearance of the larynx was healthy. The formation of the epiglottis was normal, the vocal cords were of

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Lagarde, L. Charles:—De l'Aphonie Nerveuse. [*Thèse Inaugurale* 1865.] This thesis was suggested by the researches of Dr. Krishaber, to which the author constantly appeals.

their natural whiteness : the other parts of the larynx were slightly discoloured, but with this exception presented no lesion of any kind. When the patient emitted sounds, the vocal cords were distinctly seen during deep inspiration, normally to approach or retire from each other, performing their functions in the natural physiological manner. It was observed, however, that during the emission of sounds, the vocal cords did not oscillate and vibrate upon the glottis, in so distinctly visible a manner as when the organ emits natural sounds. The patient was quite unable to utter the higher sounds.

This patient was pale, and not quite regular in her periods; but she had no symptoms of hysteria or chlorosis. Auscultation revealed nothing abnormal in the state of the lungs: the heart presented no morbid sign, except a slight clatter of the valves. All the functions were naturally performed, and hoarseness was the only symptom of which the patient complained. She positively denied having ever had any specific diseases. Examination of the external and internal genital organs disclosed no trace of antecedent lesion, except a perineal cicatrix, the consequence of a laceration during labour.

In this case, I adopted the treatment, which for so long a period I have been in the habit of employing. By means of a sponge attached to the end of a piece of whalebone, I applied a solution of sulphate of copper around the laryngeal opening. Soon after this cauterization, the voice returned.

This was evidently a case of purely nervous aphonia: during phonation, the vocal cords perfectly approached each other, and normally retired during respiration, which proved that there was neither complete nor incomplete paralysis of the inferior laryngeal nerve. Although there were no oscillatory movements of the vocal cords, or if any very slight, this was evidently the result of diminished tension of the vocal cords: there was an absence of the degree of tension indispensable to the emission of normal voice, and particularly to the articulation of the higher sounds. It appears, therefore, that in the case now under consideration there existed a functional change in the superior laryngeal nerve.

I ought to add that besides aphonia due to material lesions resulting from syphilis and tuberculosis, both morbid states produce a purely nervous aphonia.

Pulmonary phthisis, for example, may, in its last stage, produce nervous aphonia, the consequence of general exhaustion of the

system. The aphonia then shows itself with characters similar to that which is met with at the close of severe attacks of disease; and there is an extinction of voice, just as there is an extinction of all the other functions. But likewise, and without any material lesion, as is shown by examination with the laryngoscope, pulmonary tuberculisation sometimes produces nervous aphonia. Dr. Lagarde gives an example of this, which was communicated to him by Dr. Krishaber.[1]

In a phthisical young woman who had had aphonia for two months, the vestibule of the glottis, the aryteno-epiglottidean ligaments, the inferior vocal cords, and the margin of the ventricles of Morgagni were perfectly healthy. The vocal cords could approach each other, and yet there was no voice. There was, as I told you is the case when paralysis of the superior laryngeal nerves exists, neither vibration nor oscillation of the vocal cords. In eight days, however, the patient recovered her voice, without any special treatment having been adopted. She died two months later from the progress of the pulmonary phthisis. She retained her voice in its integrity to the very last, which is a proof that her aphonia was purely nervous. The laryngoscopic examination probably had some beneficial influence in re-establishing the voice in this case.

Syphilis also, without there being any lesion, may cause aphonia. To this variety of syphilitic aphonia, Diday gives the name of *secondary aphonia*, to distinguish it from that which is met with in inveterate syphilis.[2] This kind of aphonia supervenes between the third and sixth month from the appearance of the first symptoms. It commences without pain or precursory symptoms. There is at first less fulness of voice, and the alteration afterwards proceeds gradually till there is complete aphonia. Nevertheless, there is neither cough, dyspnœa, nor any general febrile state. Diday has observed these symptoms both in male and female singers affected with syphilis. In such cases, fatigue of the organ is probably the exciting cause.

Under specific treatment, recovery sometimes takes place in less than eight days. Hence, with Diday, we may ask, whether the affection is not simply a nervous disturbance of the functions of the larynx. In an absolutely similar case observed at the Hôtel-Dieu

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[1] Lagarde :—Op. cit.

[2] Diday :—*Gazette Médicale de Lyon*. T. XII, p. 35.

by Dr. Krishaber, and in which the voice returned at the end of eight days of mercurial treatment, there was no lesion discoverable by means of the laryngoscope. The case, therefore, was one of syphilitic nervous aphonia.

Nervous aphonia is not uncommon in women suffering from disorder of the menstrual function. Such a case you had an opportunity of observing in a young woman who occupied bed No. 31 of St. Bernard's ward—a case the history of which I shall by and by narrate.

In relation to the subject of purely nervous aphonia let me recall to your recollection a girl of eighteen who came into St. Bernard's ward in December 1859. Some months before admission, consequent upon a great fright, she suddenly lost her voice, and at the end of six weeks regained it, without having had any treatment. She had been ill for fifteen days, when she entered the hospital. She was working in a shop in the basement storey, on a level with the street, when all at once a cart, with tremendous noise, smashed in the shop-front. The girl, struck with terror, fainted, and had a nervous attack : on regaining consciousness, she was voiceless. On the day after her admission, in presence of all the students, I cauterised the upper part of the larynx, with a saturated solution of the sulphate of copper : the voice immediately returned. Next morning, however, as there was still some hoarseness, I repeated the cauterization, and the result was a radical cure.

During 1862, you saw three young women enter our clinical wards, suffering from aphonia; one of them had been thus affected for two months. In all three, cauterization practised, in your presence at the visit, almost completely re-established the voice within a few minutes; and after four or five cauterizations, the functions of the larynx were restored to a perfectly satisfactory state.

In June 1863, you saw a girl of sixteen occupying bed No. 16 of St. Bernard's ward. She is the patient to whom I have just alluded when speaking of the relation of aphonia to disorder of the menstrual function. When admitted, she had menorrhagic fever, accompanied by very acute uterine pains. Menstruation was easily re-established, but aphonia supervened and lasted ten days, without being in any degree modified by the different therapeutic measures which I employed. In your presence, I cauterised the upper part of the larynx with a saturated solution of sulphate of copper, applied by means of

a sponge attached to the end of a long piece of bent whalebone. The voice at once regained somewhat of its natural tone, and after the third cauterization was quite restored.

In this case, laryngoscopic examination did not disclose the existence of any local lesion. However, in some cases, though the vocal cords do not in their whole extent approach one another during the emission of high sounds, there is apparently paralysis of their tensor muscles; and in that condition, cauterization would seem to act as an excitant of muscular action. In this class of cases, Dr. Krishaber has sometimes been able to re-establish the voice by the introduction of the laryngoscope, as if a merely mechanical excitant sufficed to induce reflex action.

When the laryngoscope discloses signs of acute inflammation of one or both vocal cords, or still more, when it shows that there is ulceration, the treatment which I have just been recommending, though efficacious, is not so marvellously powerful as you have often seen it in my hands: and frequently, when two cases have been seemingly identical, the revelations of the laryngoscope have explained why the result was not so satisfactory in one as in the other. Gentlemen, I cannot too earnestly recommend you to learn how to use the laryngoscope. But I must now return to my subject.

It sometimes happens that individuals suddenly lose their voice after taking a cold bath, or after passing abruptly from one temperature to another. Sudden aphonia may also show itself consecutively to the suppression of a customary sanguineous flux; and one of the most common of this class of causes is suppression of the menses.

The remarkable sympathy which exists between the genital and vocal organs is sometimes exhibited by the occurrence of aphonia during pregnancy, after delivery, and in a still more general manner in the course of diseases of the organs of generation, especially in women, though the same remark is to a certain extent applicable to the male sex. It is in these different kinds of aphonia that cauterization at the lower part of the pharynx and upper part of the larynx has rendered me such unquestionable services, after the total failure of all previous treatment. The preparations which I employ are a saturated solution of the sulphate of copper, or a solution of nitrate of silver in the proportion of one part of the salt to five of water (by weight). The apparatus which I use for applying the solution is a piece of whalebone armed with a sponge, such as all of you

have seen me employ in cauterising the throat in diphtheria. I need not, therefore, now describe this little apparatus to you, which, moreover, as you all know, is exceedingly simple; neither is it necessary for me to-day to repeat remarks which I formerly made on the harmlessness of the operation, seeing that it is now an operation within the ordinary domain of medical practice.

The efficacy of this method of treatment may lead one to think that in these cases of aphonia, the mucous membrane of the larynx was the seat of slight inflammatory action, even though evidences of no such inflammation could be shown to exist; for in the cases which I have observed, there was neither pain, swelling nor difficulty of breathing. But the beneficial effects of cauterization of the upper part of the larynx may be explained by supposing that it produces a peculiar modality on the entire nervous system, and in particular on the nerves of the vocal apparatus. It causes a cessation of the spasm upon which the aphonia depends.

However it may be explained, this sort of cauterization is still more useful in some cases in which the existence of inflammation is very obvious, and particularly in syphilitic laryngitis in which there is no ulceration. The inflammation is characterised by pain in swallowing, in inhaling cold air, or in making an effort to speak.

The efficacy of the treatment I am now recommending, and the rapid manner in which recovery takes place under it, seem to occur exactly in proportion to the superficiality of the inflammation, if one may be allowed to employ so incorrect an expression. In aphonia supervening as the sequel of a moral emotion, or as the result of abrupt stoppage of the menses, as well as in aphonia occurring during pregnancy or after delivery, one or two cauterizations generally suffice: and you have witnessed with what rapidity recovery took place after the first operation, in our two young women of St. Bernard's ward.

I have elsewhere pointed out to you that Mr. Green of New York not only applies the caustic to the entrance of the larynx, but even introduces into the cavity of the organ the little apparatus consisting of a sponge attached to the extremity of a piece of whalebone suitably bent.

As for myself, I often employ, for the same purpose, the instrument for applying caustic invented by Dr. Loiseau, which I described when lecturing on the topical treatment of diphtheria: it is

much more reliable than Mr. Green's apparatus.[1] Prior to the discovery of the laryngoscope, before the numerous useful applications of this instrument to the diagnosis and treatment of diseases of the larynx,[2] there was no way of reaching beyond the vocal cords and to attain even so far was attended by uncertainty and many difficulties. Now, however, it has become easy to see the lesions, and by practice one can very soon acquire the art of introducing surgical instruments and medicinal appliances into the larynx.

Recovery is slower in the cases in which aphonia comes on gradually as a consequence of compulsory or excessive exercise of the voice. But aphonia associated with palpable inflammation of the larynx yields still more slowly.

One remark more before I conclude! It might be supposed *à priori*, that inasmuch as aphonia can be easily cured by cauterization, it would be much more easy to cure a mere alteration of the voice characterised by impossibility of producing certain sounds: but experience has taught us that, on the contrary, it is more easy and more satisfactory to treat complete than incomplete aphonia.

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[1] Loiseau:—*Bulletin de l'Académie Impériale de Médecine.* T. XXII, p. 1138. Paris, 1857.

[2] Czermak:—Du Laryngoscope et de son Emploi en Physiologie et en Médecine. Paris, 1860.

LECTURE XXVIII.

DILATATION OF THE BRONCHI AND BRONCHORRHŒA.

Extreme Difficulty of Diagnosis.—Dilatation of the Bronchi may be mistaken for Pulmonary Phthisis—or for Pleurisy with Perforation of the Lung. — Differential Diagnosis. — Important Signification of abundant and Fœtid Expectoration.—Causes of the Fœtor.—Dilatation of the Bronchi, unless it be to a very great degree, is not a Serious Affection.—Treatment of Bronchorrhœa, or Pulmonary Blenorrhagia.—Balsams.—Arsenical Inhalation.

Gentlemen :—The facts observed in early life are those which are the most permanently engraven on the memory : and very often, now that I am approaching old age, I recollect the most minute circumstances connected with cases of which I took down notes when on the threshold of my professional career.

In 1823, when I was still an hospital pupil, my excellent master Bretonneau had under his care a Parisian architect, who had long been an invalid, and had been recommended to go to the Eaux-Bonnes by his medical attendant. At that period, there were no railways : the patient consequently posted, travelling by short daily journeys. The first halt was at Orléans, and the second at Tours. On arriving at Tours, he was so exceedingly fatigued as to be unable to proceed. Under these circumstances, he sent for Bretonneau.

The appearance of the patient seemed to tell pretty plainly the nature of the disease under which he was labouring. Frightful emaciation, a yellow clayey complexion, continued fever, night sweats, and very copious muco-puriform expectoration, were the symptoms which he presented. Bretonneau felt almost certain that the disease was tubercular phthisis. Auscultation, however—then a recent art, which Bretonneau had studied with great enthusiasm—

did not furnish the signs usually met with in phthisical persons. He neither found a dull sound on percussing the upper part of either lung, nor did he hear, as he expected, gurgling in one of the superior lobes. The patient died within a few days; and at the autopsy, made with the greatest possible care, no trace of tubercle was discovered: Bretonneau recognised chronic inflammation of the bronchial mucous membrane; but he did not examine the tubes with a view to ascertain whether they were dilated in some places, or whether they were throughout of normal calibre. It must be stated that in those days, attention had not as yet been so much directed to the symptoms of dilatation of the bronchi, as it was in 1825, when Laennec published the second edition of his immortal treatise on auscultation.

Laennec's description of dilatation of the bronchi is complete, although it was probably thrown off at the first dash. New facts have been added by the researches of Dr. Barth; but he confirms, in almost every particular, the previous statements of the illustrious physician of the Necker Hospital.

If you read the cases narrated by Laennec—especially his fourth case, to which he seems to attach most importance—you will become convinced that it is exceedingly difficult to diagnose between phthisis and bronchial dilatation, by observing that the illustrious founder of the art of auscultation hesitated, and, up to the autopsy even, remained in doubt: then too, will you be better able to understand how Bretonneau's diagnosis was at fault in the case which I have just related to you.

The first case in the excellent monograph of Dr. Barth affords testimony to the same effect. In 1835, Drs. Louis and Barth saw a woman die in their wards, whom both supposed to have had tubercular phthisis in the third stage, and yet at the autopsy, it was seen, that the tubercular lesions were quite unimportant, and had no possible relation to the very formidable symptoms which had terminated in death: but they found enormous bronchial dilatations. Most probably, the morbid condition of the Parisian architect would have been proved to have been similar, had the attention of Bretonneau been directed to the subject now before us when the case came under his notice.

First of all, then, Gentlemen, it appears that certain attacks of bronchial catarrh may give rise to all the symptoms of tubercular phthisis; and I am only speaking of *symptoms*, for stethoscopic

signs are generally absent, at least in cases in which the bronchial dilatations are not confined, as is sometimes observed, to the summits, or in others, in which dilatations exist both in the upper and central parts of the lungs: in such cases, diagnosis is nearly impossible.

There is still another source of error, which has to be added to those I have mentioned. It sometimes happens that patients during the course of the catarrhal affection are seized with hemoptysis; and in place of quoting numerous examples, let me refer to Laennec's well-known case (Case 4th), in which upon two occasions the patient had spit blood within six weeks of his admission to the Hôpital de la Charité, and to the seven similar cases which constitute the basis of Dr. Barth's memoir. A perfectly similar case, which has recently occurred in my own practice, I shall immediately bring under your notice.

If you consider, that according to the testimony of Dr. Barth, bronchial dilatation exists on one side only in the majority of cases, that it as frequently occupies the summit as the base of the lung, and that the disease, when extensive, is very often accompanied by hectic fever, muco-purulent expectoration, and nearly all the symptoms of consumption, you will be a little more indulgent to those who make an erroneous diagnosis in such cases.

It is true, Gentlemen, that errors of this kind are not of very great importance; for although we may sometimes be able to intervene usefully in cases of bronchial dilatation, the treatment does not materially differ from that usually instituted in tubercular phthisis. In fact, the therapeutic indications are the same; such as moderating, as far as is practicable, the catarrhal flux, the sweating, and the fever, sustaining the flagging vital powers, and, in a word, contending against the conditions more immediately dangerous to life which arise, leaving alone, as of secondary importance, the lesions against which our resources are impotent.

Gentlemen, the remarks now made are not offered as the preamble of a bill of indemnity which I ask from you in respect of the young woman we have just lost in St. Bernard's ward, whose case was one of the most remarkable examples of bronchial dilatation which I have had an opportunity of observing. The diagnosis, established with precision on the day of admission, has been confirmed at the autopsy. I will admit, however, that sometimes my confidence in my diagnosis was shaken; and when the symptoms of a hectic

condition became more decided, and when the fœtor of the sputa increased, I became afraid that I had committed a mistake: several times, I hesitated in my diagnosis between bronchial dilatation and pleural effusion communicating with the bronchi by a pulmonary perforation; but I reverted to my original opinion, thus giving you a specimen of uncertainty which would have been much greater had the principal lesion occupied the apex in place of the central and inferior portion of the lung. I shall now give you a summary of the history of this case.

The patient was a woman of thirty years of age, little, thin, and puny, who, on 2nd June 1863, came to occupy bed No. 6 in St. Bernard's ward. She had had a cough from her earliest infancy; and although she had never had an attack of asthma, was habitually out of breath. The menstrual function was normal, she had never had hemoptysis, and no member of her family had had tubercular disease. She had had frequent attacks of inflammation of the chest, accompanied by severe stitches in the side. She had been confined twenty-one months before admission to hospital, and, till within a month, had nursed her infant. From that time, her cough increased: she had had constant fever for a fortnight, but till then, was able to attend to her household duties.

This woman, as I have said, was thin: she had curving in of the nails—hippocratic nails, as they are called—and yet her countenance was not that of a phthisical subject.

Percussion below the spine of the scapula was very resonant: and the resonance was excessive at the middle and posterior part of the right lung, which was evidently dilated in that situation. Over the middle and inferior part of the left lung there existed, on the contrary, flattening of the thoracic walls, and well-marked dulness. No vesicular expansion, no expiratory murmur, nor blowing sound could be perceived on auscultating the supra-spinal and infra-spinal fossæ. In the right subclavicular region, and there only, the expiratory murmur was slightly prolonged. But in the middle and inferior regions, along the left vertebral hollow, there were heard mucous râles combined with coarse gurgling and mucous blowing: the voice of the patient was so weak as not to be in the slightest degree resonant in that situation. Within twenty-four hours, the patient filled two or three spittoons with expectoration, which was purulent, semi-opake, semi-salivary, somewhat frothy, and of a sickly, almost fœtid odour. This fluid was brought up by an effort

to vomit rather than by expectoration, after two or three fits of cough; and at each time two or three spoonfuls were discharged.

My diagnosis was—the existence of *chronic bronchitis*, and considerable *bronchial dilatation* at the middle and lower part of the left lung, and the *absence of tubercles*. I prescribed eight turpentine capsules, and fumigations with arsenical paper.

By the 7th June, the oppression in the breathing had increased, and on that day the fever was higher than on the previous evening. Very extensive fine subcrepitant râles—the râles of acute bronchitis —were heard. An emetic dose of ipecacuan was administered with the effect of producing decided relief.

Five days later, remarkable fœtor of the breath was perceived, although there was not a corresponding fœtor of the sputa. The pulse was 124, and the respiration 48 in the minute. There was orthopnœa. Over the right side generally, fine mucous râles were heard. An attack of acute bronchitis had evidently become chronic.

On the 12th June, at the evening visit, the pulse was 128: the respiration, which was very anxious, remained at 48: the skin was dry, and burning. There was pain on both sides of the chest. On auscultation, fine mucous râles were heard posteriorly throughout the whole of the right side: the râles were nearly crepitant, and dry at the base. On the left side, down to the middle third of the lung, there was gurgling along with blowing, and a somewhat amphoric sound of the voice, while fine mucous râles were audible at the base. Dry cupping with eight glasses, and an ipecacuan emetic afforded remarkable and almost immediate relief.

Next morning, the fine râles, so distinctly heard on the previous evening, were no longer audible. There were no longer any vibrating râles. But in the evening, the fine mucous râles returned, and the respiration again became anxious. The ipecacuan was repeated, but not with the same success as on the former occasion.

On the 15th, there was no increase in the frequency of the respirations, but the pulse was 140, at which it afterwards remained.

On the 17th, râles, almost cavernous, were perceived at the external angle of the left scapula in the subspinal fossa: some of them almost had an imperfect sound of metallic tinkling. No change had taken place, in the patient's general state, which was very

bad. Slight perspiration was visible on the forehead and front of the chest.

On the 19th, there was slight sweating. The countenance was greatly changed, and had a pale bistre colour. The voice was plaintive and feeble, but not extinct. The sputa, which had become as fœtid as the breath, surged up in large quantities, filling four or five spittoons daily.

On the 20th, the pulse was 148 ; and the respiration only 44.

On the 22nd, the face changed, the features becoming expressionless, the naso-labial groove deepening, and everything announcing that the end was near.

On the 24th, death occurred.

It is worthy of remark that this woman, who in the Grecian meaning of the term was phthisical, had never had the aspect of a person affected with tuberculosis, and had had neither colliquitative sweats nor colliquitative diarrhœa. We have just seen that the progressive wasting of the body and death necessarily resulted from the progressive and continuous diminution of the sources of hematosis, and the enormous extent of the daily loss by bronchial suppuration. It may be asked whether pregnancy and prolonged lactation did not produce in this case of chronic bronchitis the same baneful influence which they cause in phthisis.

The following is an account of the anatomical lesions met with in this case. The lungs were voluminous and very heavy ; they did not collapse when the chest was opened. They were closely bound, particularly on the left side, where the pleura had nearly disappeared, to the parietal pleura, by very numerous adhesions. Similar adhesions united both pleuræ at the part corresponding to the pericardium. There was no effusion into the pleural cavities. The adhesions were evidently the remains of the numerous attacks of pleurisy described by the patient when giving the historical details of her malady. The right lung, emphysematous throughout nearly the whole of its extent, was solidified in many places to such an extent that, notwithstanding the emphysema of which it was the seat, the pulmonary tissue presented a remarkable consistence. At the lateral surface of the inferior lobe of the right lung, a cavity was found capable of holding a small hazel-nut : its walls were soft, pultaceous, of a yellowish grey, and yielded a gangrenous odour.

Perhaps this explains why the breath was more fœtid than the sputa. Around the excavation, there was no tubercular deposit, but

the surrounding parenchyma, which presented a blackish red colour for about five millimeters, had a density nearly equal to that of hepatisation. Upon one of its walls, there opened the orifice of a small dilated bronchial tube. This small cavity was evidently not of tubercular origin, but the result of a process simultaneously inflammatory and gangrenous. There was no tubercular deposit at the apex, where the lung was exceedingly emphysematous: it was slightly vascular and immediately collapsed when cut into, as is the case in vesicular emphysema. When an incision was made into the pulmonary tissue, a liquid exuded similar to that which the patient ejected during life. The lower portion of the superior lobe presented incipient hepatisation. The second and third divisions of the bronchi were much dilated: their mucous lining was injected and of a somewhat slaty colour.

The left lung had a solidified appearance, especially its posterior aspect, which was red and marbled: on moving the finger, however, over the middle part of the surface of this lung, places were met with which were very easily depressed, and were really caverns corresponding to the situations in which gurgling had been heard during life. There were about twelve of these cavities varying in capacity from the size of an almond to that of a walnut, filled with a whitish, cheesy substance, apparently concrete pus. The membrane lining these cavities, so far from having the thickness and hardness belonging to tubercular cavities, was exceedingly thin. There opened into one of them a small bronchial tube, which was dilated throughout its whole extent. The pulmonary tissue intervening between the cavities presented the appearance of mere plates of conjunctival tissue, bloodless, almost transparent, and seemingly devoid of contractility. On making a section of the lung, through the assemblage of cavities, the appearance presented was that of a cut sponge, or to employ a still better comparison, of the lung of a batrachian reptile. Some of the cavities communicated with each other, and were only separated by small imperfect partitions, resembling the valves of veins both in form and slightness.

This alteration of texture has been specially described by Laennec. The presence of these cavities near the surface of the middle of the lung explained the gurgling heard during life, on auscultation; and the large quantity of dense cheesy matter which they contained accounted for the dulness on percussion.

Nearly all the bronchial tubes were dilated; but one, of the second

order in respect of calibre, going to the inferior lobe, was specially remarkable, from exhibiting about its middle an ampullary dilatation in diameter equal to that of the great bronchus: in the situation of this protuberance, the mucous membrane had a violet red colour. Most of the tubes which opened into the cavities already described as containing semi-concrete purulent matter were divisions of this enlarged tube.

The superior lobe of this lung was a typical illustration of vesicular emphysema: it had a greyish white colour, gave to the touch the sensation communicated by a down pillow, and collapsed when cut. Like the superior lobe of the right lung, it contained no tubercle. At the lower part of this lobe, there were seven or eight cavities similar to those so numerous at the middle and lower part of the inferior lobe of the same lung.

The bronchial glands were very large; when cut, they showed a blackish grey appearance; and there was no trace of tubercle.

To sum up the description:—there was vesicular emphysema of the upper part of both lungs; bronchial dilatation, and numerous cavities particularly in the middle and lower portions of the left lung; here and there, hepatisation; and nowhere, any tubercle. Such were the structural changes in the organs of hematosis in this patient, who scarcely breathed, except with the upper parts of her lungs—and these parts were emphysematous!

You have all been struck with the extreme fœtor of the breath in this case, which was almost intolerable when the patient coughed, and constituted a great source of annoyance to those who occupied adjoining beds; and yet it did not taint the expectoration. The sputa were diffluent, muco-puriform, and exceedingly copious, the quantity brought up in a day being at least a litre (rather more than an Imperial British quart); but their sickly and somewhat nauseous odour fell far short of the breath in disgusting fœtor.

There are two points of importance to consider in reference to the expectoration—its great quantity and its fœtor. I wish to discuss with you the great diagnostic value of both.

Extreme fœtor of the breath is observed in gangrene of the lung, and occasionally in tubercular phthisis, but in phthisis it is generally transient, seldom lasting more than three or four days: in gangrene of the lung it certainly continues longer, particularly in that strange kind which attacks many lobules in succession; but in such cases, it is very powerful for some days, when it moderates and then returns

as it was before, and again ceases to be powerful: these alternations, without the aid of any other signs, are quite sufficient to guide the practitioner to a correct diagnosis. There is something distinctive in the odour of gangrene: and in bronchorrhœa connected with dilated tubes, the smell is quite different, being suggestive of the presence of putrescent animal matter.

I am aware that in successive lobular gangrene of the lung, the duration of the fœtor may be considerable; and I recollect its continuing for nearly three months in a lady who was under the care of my accomplished friend Dr. Lasègue and me. But in bronchorrhœa connected with bronchial dilatation the offensive smell continues for a very much longer period.

In 1848, I saw in the rue St. Honoré, Paris, along with my honourable friend Dr. Louis, a man between sixty-two and sixty-three years of age, who had bronchial catarrh and dilatation of the bronchi. At the time when we were sent for to this patient, he had been seriously ill for several months, and after we had attended him for two months without his being benefited, he sought other advice. During the entire course of the disease, the smell of the breath was such as to render pestiferous the whole of his suit of rooms; and even the staircase leading to them was redolent of the same stench. I did not know what had become of the patient, I believed him to be dead, when, in May 1863, that is, fifteen years later, I was sent for to see one of his daughters, from whom I learned that he was still alive, and still had a bronchial cough, which, however, except obstinacy, had no peculiar character.

The persistence of fœtor, when there is nothing else to lead to the belief that lobular gangrene of the lung exists, is in itself an important diagnostic sign of dilatation of the bronchi.

Nevertheless, Gentlemen, it may happen that for several months the expectoration is copious and fœtid, although there is only a simple pulmonary catarrh: in some persons a common bronchial flux leads to consequences similar to those induced by certain fluxes connected with inflammation of a mucous membrane. It was the other day only, when speaking to you of ozæna, that I mentioned that in both sexes the gonorrhœal discharge sometimes assumes an extreme degree of fœtor, and also the flux in acute and subacute coryza, in circumstances in which it is impossible to assign the cause of this; and moreover, this stench is not always met with in the same individuals although placed under apparently identical con-

ditions. In certain epidemics of influenza, or under the influence of the herpetic diathesis, for example, the bronchial flux in some persons acquires an extraordinary stench which will continue during the continuance of the special phlegmasia on which the flux depends. This is perhaps the very thing which occurred in the patient mentioned to you in whom Dr. Louis and I suspected dilatation of the bronchial tubes, and who fifteen years subsequent to our forming this diagnosis was enjoying such good health as to lead us to suppose that we had erred in diagnosis; for it rarely occurs that bronchial dilatation diminishes as age increases.

Gentlemen, if for several months, the expired air is continuously fœtid, it is a diagnostic sign of great value in bronchial dilatation: copious expectoration is not a less valuable sign. You have seen how much importance I attached to this sign, and how it has imparted confidence to my diagnosis. The diffluence and extreme copiousness of the sputa are hardly ever found except in cases of pleural vomica, unless we have to do with bronchial dilatation. Sometimes in the case before us, you have seen me hesitate, particularly when, during the patient's efforts to cough, the gurgling assumed the sound of metallic tinkling: nevertheless, I was brought back, in spite of myself, to my original diagnosis by the following special consideration. Undoubtedly, when a collection of fluid in the pleura makes a way for itself into the bronchial tubes, a diffluent and very copious expectoration supervenes: but in such a case, the copiousness is sudden, on the day after its occurrence, it decreases, and although the quantity of matter brought up continues to be considerable for some days, after the lapse of that time, the copiousness is never so great as when the perforation took place, unless indeed there be hydro-pneumothorax, in which case enormous quantities of matter may for weeks continue to be discharged.

Here, however, Gentlemen, there is not much danger of confusion. The signs of hydro-pneumothorax, when the cavity is of considerable size, are unmistakeable even by a somewhat careless physician; and when the morbid cavity is much circumscribed, the quantity of the flux is also much limited. You will recollect that when I was hesitating between belief in a pleuro-pulmonic perforation and a dilatation of the bronchi, I was always brought back to the latter view by the fact that I could never hear metallic tinkling, hippocratic gurgling, nor tympanitic resonance in any part of the chest.

I am well aware that a sign existed which greatly shook your confidence—I refer to the presence of dulness posteriorly of the affected side of the chest. This dulness which has been explained to you, not by condensation of the lung as pointed out by Laennec (and as is the general cause of dulness), but by the presence of an enormous quantity of semi-concrete matter in the ampullary cavities—this dulness I say, was not so complete in our case, and indeed never is so complete, as in pleurisy. But I quite understand that it may lead to an error in diagnosis, that it may lead to the belief that pleurisy exists, and so to the conclusion that there is a communication between the cavity of the pleura and the bronchial tubes.

Gentlemen, I have no intention of giving you in this lecture a complete account of dilatation of the bronchial tubes, a subject which you will find so well explained by Laennec, and by the later researches of Dr. Barth; but I was unwilling to allow the case which has been engaging our attention to-day to pass without pointing out all its clinical importance, and without making you aware of the magnitude of the difficulties with which the diagnosis of bronchial dilatation is surrounded.

The lung which I showed you on the anatomical table is an example of bronchial dilatation in its extreme degree; and I do not believe that any case is on record in which this lesion has been found more extensive. Our patient was actually disfigured, so to speak, by the great extent of the structural alterations; and you would form a very erroneous idea of the affection were you to regard the lung of which I am speaking, as a typical specimen of bronchial dilatation.

Chronic bronchitis usually, and to a certain extent necessarily, causes vesicular emphysema. The vesicles and bronchial tubes, however, give way more easily in some subjects than in others: in the majority of cases, the vesicles become dilated, and the intervesicular tissue becomes condensed: the dilatation of the vesicles may proceed to such a degree as to cause their rupture, whence originate the large vesicles which sometimes give to the human lung the appearance of the lung of a batrachian reptile, as was seen in so remarkable a degree in the case of our patient. In comparing the lungs of patients presenting a very great degree of vesicular emphysema with lungs which are quite free from disease, a certain amount of attention only is required to perceive a fact which at a first glance might escape

notice, viz. that besides the vesicular expansion, there is dilatation of the trachea and bronchial tubes: from the uniformity of the dilatation, it is the more apt to escape notice. The same remark is applicable to vesicular dilatation, when it is everywhere in the same degree: a certain amount of attention is then necessary to detect it, although the general enlargement of the lung, and its not collapsing, testify to the existence of the lesion. Bronchial dilatation may be, and in fact ought to be, regarded as an emphysema of the bronchial tubes. In most cases, it is equally distributed, and associated with vesicular emphysema: in other cases, it is unequally distributed, and then constitutes the affection known as *bronchial dilatation*, in which the dilated bronchi bulge out either into moniliform protuberances, having exactly the appearance of a string of beads, or, as is more usual, expand into elongated, fusiform, or ampullary shapes, as in the case now before us. Many of the bronchial cavities may communicate with one another, so as to give the lung the appearance of collections of united abscesses, or still more the appearance of certain multilocular ovarian cysts after they have been cut open and had their fluid contents evacuated. At the same time it can be seen, that the pulmonary tissue between the large cavities is condensed, a condition which explains the frequency of dulness on percussion, a common sign of extreme bronchial dilatation.

I have often asked myself when looking at this induration of the lung, and at the evidences of chronic pleurisy so commonly found at the autopsy, whether the large cavities in the pulmonary tissue were not real vomicæ formed by the softening of the inflamed, suppurating lobules. When we come to study the lobular pneumonia of children, we shall see that it is very usual in that disease to find purulent collections of the size of a millet seed or a lentil, or, exceptionally, as large as a small cherry. It is usual to admit in these cases—and as for myself I give my formal adhesion to the doctrine—that there has been an inflammatory melting down of a union of lobules, and the opening into the bronchial tubes of the little abscesses so formed. It is believed that the inflamed pulmonary lobule passes through all the degrees of hepatisation to the third stage, to the stage of purulent softening; and it is asked whether something analogous may not take place in the adult, in some cases of bronchial catarrh. The case now under review seems to indicate an affirmative answer to this inquiry: indeed, in some places, the lung has a

greenish black appearance, and is evidently sphacelated. In bronchial dilatation, therefore, there are several degrees: there is that degree in which the air tubes are dilated, and to which the term bronchial dilatation is properly applied; while there is another degree in which lobules or parts of lobules are destroyed by compression or by the mere inflammatory process itself, which may produce ampullary cavities, hardly resembling bronchial dilatations it is true, and being more like purulent cavities than dilatations.

Whatever there may be in this opinion, an opinion which I would not venture to maintain positively, and which rests on an examination of the pathological anatomy of the parts, there still remains one peculiarity in this disease which I wish to point out and briefly illustrate. On reading the different cases which have been published, one is struck with the apparent harmlessness of the disease until it has nearly reached its last stage. The young woman with whose autopsy we are now engaged was not really ill till within a month of her death, for up to that period, though in precarious health, she went about her usual occupations. There was nothing in her condition to justify the belief that her end was so near. The aggravation of the symptoms came on rather suddenly, as also occurred in many of the patients whose histories have been related by Laennec and Barth. The celebrated subject of Laennec's *fourth case* did not discontinue work till within a few days of admission to hospital. Bronchial dilatation in itself, then, is only of secondary importance in respect of danger. Indeed, if you reflect upon the circumstance, that the local lesion is often so slight as to be limited to only one bronchial ramification, and extends sometimes only to as many tubes as in the aggregate do not constitute the hundredth part of the respiratory area, you will admit that occasionally it must be very difficult during life, to detect bronchial dilatation, particularly when there is a total absence of general symptoms.

On July 2nd, 1863, I received a patient, about sixty years of age, in my consulting room. He came to consult me about a catarrh accompanied by oppression of breathing, from which he had suffered for more than two years. During the hour which he had to wait his turn for consultation, he filled a pocket handkerchief with copious diffluent sputa. He stated that he had often had slight hæmoptysis, and that occasionally the expectoration became very fœtid. He said that he had not had fever, and that his general state of health was

not bad. I suspected that there was bronchial dilatation; and on examining the chest, I obtained results which I shall now state. On the right side, there were signs of vesicular emphysema; on the left side, the lower half of the chest was to some extent flattened, and there was much less than the normal amount of resonance. On auscultation, I heard coarse gurgling, with suction sound and vocal resonance, similar to that which is so often observed at the summit of the lung in tuberculous subjects.

The point in this case, Gentlemen, to which I wish to direct your attention is the following:—The patient walked, followed his usual occupations, and had no fever: although he had considerable bronchial dilatation, he did not suffer much, and his state was endurable, though he always had oppressed breathing and copious expectoration.

When the dilatation is slight, it can hardly be called a complication, as it does not increase the danger of the bronchitis: but if it extend to an entire lung, and still more, if it extend to both, there is real danger, the causes of which are easily understood. First of all, the patient has available for the requirements of hematosis, only three-fourths, the half, or two-fifths of the pulmonary parenchyma. If, under such circumstances, an attack of bronchitis or pneumonia supervene, and respiration is without an apparatus, the patient necessarily dies. On the other hand, when we observe that a lung is in a very advanced state of bronchial dilatation, we have reason to believe that the pulmonary parenchyma surrounding the dilated tubes is the seat of chronic inflammation, which, under the influence of even slightly irritating causes, will become subacute.

There is still another cause of danger which I cannot pass over in silence: it is one which was apparent in the young woman whose case we are now considering. You saw the enormous pouches containing accumulations of semi-concrete pus, in appearance very like putty, and exhaling a frightful stench. I do not wish to affirm positively that the putrid discharge in contact with the diseased surfaces, and floating in the bronchi, carried by successive inspirations into the air-passages leading to healthy portions of the lungs, may not become a source of infection to the whole economy, a source all the more prolific, that the respiratory surfaces are of all parts of the body those which absorb with the greatest ease and rapidity, as is shown by the phenomena of respiration, by the sudden

overwhelming effects produced by the inhalation of ether, chloroform, and some deleterious gases.

Upon the whole, Gentlemen, dilatation of the bronchial tubes is only a consequence, and one of the forms, of chronic bronchitis. I bring it prominently under your notice, because it presents symptoms and stethoscopic signs which deserve a little special attention.

When bronchial dilatation has attained the point at which we found it in our patient, there is generally little to be done, and all our attempts are failures; but in the more usual form of the affection, the symptoms improve, and even disappear, when the bronchitis is cured. The fever ceases, the flux diminishes daily, and nothing remains of the affection, except habitual expectoration in the morning, which does not seem to affect the health.

The treatment is similar to that which is employed in common pulmonary catarrh. In the acute stage, emetics are given: if there be high fever, digitalis is useful: when the oppression is urgent, ammoniacum, powerful remedies of the family *solaneæ*, and the fumes of nitre are indicated; sometimes, we must rely on cutaneous revulsive measures, such as smearing with tincture of iodine, the use of carrot poultices, frictions with croton oil, and the application of flying blisters. But if, as often happens, the flux is excessive in quantity, there are new indications for treatment after the acute stage is over: they pertain to the *bronchorrhœa* or *pulmonary blennorrhagia*, regarding which I now proceed to make some remarks.

In the case of a patient occupying bed No. 13 of St. Bernard's ward, you heard me prescribe a potion of balsam of copaiba for chronic bronchial catarrh with copious mucous secretion, a form of catarrh which I have called *pulmonary blennorrhagia*. I must state my reasons for thus speaking and acting.

Without at all instituting a forced analogy, we may say that catarrh of the air-passages, at least when accompanied by abundant mucous flux, admits of comparison with the catarrhal affections of the genito-urinary organs to which we give the name of *blennorrhagia*. Recollect that there are different kinds of blennorrhagia.

There is one kind of blennorrhagia to which the name is specially applied, and the specific character of which no one will dispute: it is a contagious venereal catarrh contracted by sexual intercourse with a person who has the affection.

But independent of simple venereal blennorrhagia, there is a

form which is symptomatic of chancre in the urethra. It is *syphilitic*, and is a distinct species of blennorrhagia.

Along with these two species of blennorrhagia, there is another, which supervenes under the influence of connection with a menstruous woman, or with a woman who has leucorrhœa. This species is much rarer than some medical men believe, and much rarer than many patients allege.

Ozanam, Blas of Madgebourg, and other trustworthy authors have described *epidemics of blennorrhagia* occurring under certain medical conditions of the atmosphere, in which the discharge lasted for some days, and then, as a general rule, ceased spontaneously.

Cases of *rheumatic blennorrhagia* are likewise recorded. It occurs in persons subject to arthritic pains, and in whom their sudden cessation has been followed by the appearance of a urethral discharge; or the reverse may occur, and the sudden stoppage of the urethral discharge may be followed by a return of the arthritic manifestations of rheumatism. In gout, this occurrence is still more common.

Swediaur recognised herpetic blennorrhagia: it is a species which perhaps is allied to the gouty. It is common in women, and rather rare in men.

The *influence of diathesis* upon the production, form, and progress of blennorrhagia is a subject which has recently been taken up anew and ably argued by my pupil and friend, Dr. Peter. In a discussion which he raised in the Medical Society of the Hospitals of Paris, this physician maintained that blennorrhagia is not univocal; that it appears and recurs most readily in persons of rheumatic, gouty, herpetic, or scrofulous diathesis; that in such persons, it takes from the existing diathesis its specific characters, and consequently that along with, and in complement to, the topical treatment of the urethra, recourse must be had to the remedies which have been found useful in the cure of gout, herpes, and scrofula. Dr. Peter is far from supposing that when arthritis or ophthalmia supervenes during the course of an attack of blennorrhagia, we have to do with *blennorrhagic* rheumatism or ophthalmia; and he considers it more correct to say, that there is *rheumatic* blennorrhagia, arthritis, or ophthalmia. He considers that the arthritic diathesis gives rise to all the complications, the blennorrhagia being only the exciting cause. The blennorrhagia itself could not be produced unless the diathesis existed.

To this doctrine, which is essentially medical, I give my adhesion. It explains the failure which in certain cases attends the treatment blindly followed by specialists, and opens up therapeutic plans full of resources.[1]

Hunter pointed out that among the complications which follow in the train of *difficult dentition*, there occurs a purulent discharge from the penis accompanied by difficult and painful micturition, and exactly simulating a violent gonorrhœa.

Some *fermented drinks*, particularly beer, when taken in too great quantity, are causes of blennorrhagia; and every body admits the distinction which exists between this species of urethral catarrh, and those other forms of which I have just been speaking.

Finally, let me remind you that blennorrhagia is also sometimes the result of mechanical irritation of the penis, masturbation, or venereal excess, as well as of the other causes which I have mentioned. And to the same category belongs the blennorrhagia so frequently the sequel of the introduction into the urethra, or the prolonged continuance therein, of a sound.

In applying the term blennorrhagia to muco-purulent catarrhal secretions from the surface of mucous membranes, from the mucous membrane of the eye for example, it is necessary to distinguish different species, just as in blennorrhagia from the genito-urinary organs.

A child in coming into the world contracts a purulent ophthalmia from its mother, who is the subject of vaginal blennorrhagia: that is, a case of *venereal ocular blennorrhagia*. Another infant, born during the prevalence of an epidemic of puerperal fever, will take purulent ophthalmia of puerperal character: that is a case of *puerperal ocular blennorrhagia*.

There is a third species of ocular blennorrhagia which is very different from either of the two I have already mentioned. I refer to the catarrhal ophthalmia vulgarly called "*cocotte*"—that strange epidemic ophthalmia, equally prevalent among adults and children, which is characterised by a muco-purulent discharge from the palpebral conjunctiva. It is very different from the ophthalmia produced

[1] PETER, (Michel):—De la Blennorrhagie dans ses Rapports avec les Diathèses Rhumatismale, Goutteuse, Scrofuleuse, et Herpétique. Paris, 1867.—See also the *Union Médicale* for November and December 1866, and February 1867.

by simple mechanical irritation of the mucous membrane of the eye, caused by the presence under the eyelids, of dust, snuff, or any other foreign body.

Well, then, Gentlemen, catarrhal affections of the pulmonary apparatus present an analogy to the catarrhal affections of other mucous membranes, to this extent, that in both we find different species, and that to all of them the term *blennorrhagia* is equally applicable.

Attacks of *pulmonary blennorrhagia* may arise from simple irritation of the mucous membrane, such as those which supervene under the influence of the inhalation of cold air, or the vapours of chlorine, iodine, and arsenic. The irritation, after having in the first instance given rise to a slight muco-purulent discharge, produces (when it becomes more intense) a copious blennorrhagic flux, which may be compared to those which we have seen occur in the urethra and the eye—in fact, a true pulmonary blennorrhagia.

Pulmonary blennorrhagia arises from very different causes. The cause may be the existence of that special, epidemic, and unquestionably contagious disease which we know under the name of influenza [*grippe*] : or it may be measles, which as you know is very often accompanied by a violent catarrh characterised by cough and expectoration, the sputa often being muco-puriform and so copious as to resemble the catarrhal affection in phthisis : or again, the pulmonary blennorrhagia may be a simple catarrh.

I have no intention of giving you an account of these different species of catarrh. The similarity which I have established between them and urethral blennorrhagic discharges will suffice to explain the treatment which I instituted in the case of our patient in St. Bernard's ward.

The administration of balsamic preparations in the treatment of the catarrhal affections of the genito-urinary organs in both sexes is in the present day vulgarised to such an extent, that not only is it followed by nearly all practitioners, but it is even resorted to without medical advice by the majority of persons who find themselves attacked by urethral blennorrhagia, the medicine principally employed being copaiba. Although this drug is not an infallible remedy in these cases, the frequency with which it proves really efficacious is incontestable.

A patient comes to consult you in a case of this sort : your first prescription embraces the use either of this medicine, or of turpentine, or of cubebs (which has properties similar to those of copaiba) ;

while at the same time, you order some stimulating solution to be injected. Whatever may be the nature of the urethral catarrh, your treatment is pretty nearly the same; and cure, though it may be more or less delayed, according to the species of the disease, is always the final result.

How does it happen, then, that pulmonary blennorrhagia is not more frequently treated by balsamic preparations, seeing that so much success attends their administration in urethral blennorrhagia? We are too apt to imagine that the mucous membrane of the lungs being situated more in the interior, and concealed from our sight, is consequently more beyond the reach of remedies. There is nothing in this idea; and when we have failed to act upon the affection with our remedies, it is because they have not been properly administered.

Whatever may be the species of the pulmonary blennorrhagia, whether it depend on the specific catarrhal disease called influenza, or on morbillous catarrh, herpetic catarrh, or on catarrh of some other species, it will derive real benefit from the same medicines which are appropriate for the cure of urethral blennorrhagia.

It is, however, perhaps, in cases of muco-purulent bronchorrhœa, cases in which it is not unusual to see the expectoration amount to several pounds in the twenty-four hours, with very little cough and no symptoms of irritation—such cases as are particularly common in old people—that the balsamic remedies (at the head of which I place balsam of copaiba and essence of turpentine) are most specially indicated. More than once I have met with this form of pulmonary catarrh closely simulating confirmed phthisis; and the frequency of this occurrence led physicians of old times to accord a very high value to balsamic remedies in the treatment of phthisis. It must be admitted, that in spite of all the improvements attained in our local means of diagnosing pulmonary phthisis, the symptoms of bronchorrhœal affections, usually accompanied by general or partial dilatation of the bronchial tubes, often mislead us still, not only when there is apparently a frightful amount of purulent softening progressing in the lungs, combined with the co-existence of nocturnal sweats, diarrhœa, and marasmus; but likewise, as I stated at the beginning of this lecture, in consequence of the bronchial dilatations sometimes furnishing on auscultation several of the signs looked upon as pathognomonic of the third stage of phthisis. It is proper to add, however, that in chronic catarrh, these signs are most frequently observed at the base of the lungs, whereas they are,

on the contrary, most commonly found at the upper part of the lungs when there are tubercles.

The treatment of pulmonary catarrh by balsamic preparations is far from being a novelty. Dioscorides, who perhaps only repeated a fact in therapeutics which had already been placed on record by Hippocrates, said that turpentine and the other resins purged the lungs of morbid matter. Without, however, going so far back in the history of medicine as the times of Hippocrates, you know that Morton lauded the balsams, especially the balsam of Tolu, which is one of the ingredients of his famous pills.[1]

At the beginning of this century, physicians looking to the effects obtained from the balsam of copaiba in the treatment of urethral blennorrhagia, and attaching importance to the analogy which I have now pointed out between catarrh of the lungs and catarrh of the genital organs, conceived the idea of employing it in pulmonary catarrh. Hallé has mentioned a remarkable example in which a patient suffering from chronic pulmonary catarrh with copious expectoration of purulent appearance was cured by the balsam of copaiba. More recently the American journals published wonderful results obtained by Drs. Armstrong and Laroche by the use of similar means; while at the same time, in France, Dr. Avisard was showing the efficacy of turpentine.

Gentlemen, you are acquainted with the manner in which these medicines—copaiba and turpentine—are most easily administered. To mask their disagreeable taste, they ought to be given in gelatinous capsules containing from 15 to 20 drops. Administered in this way, a patient may take from one to six grammes of either of these substances within the twenty-four hours.

The ingestion of the medicines being accomplished in this manner, they are absorbed, their active principles are carried into the circulation, and exhaled from the surface of the pulmonary mucous membrane, quite as well as from any other mucous surface. The characteristic odour of the breath of persons taking these medicines clearly indicates that this statement is correct: moreover, the same odour is apparent in their urine and fæces, showing that the balsamic substances have also been presented to the genito-urinary and intestinal emunctory organs. These remedies, then, act upon the

[1] Morton:—Phthisiologia; cap. vii—De indicationibus curativis phthiseos originalis.

different mucous membranes when affected with catarrh in such a way as to modify their condition and determine a new state—a sort of morbid irritation—which brings to an end the pathological state, the morbid irritation of which they were the seat. Here, we have a substitutive treatment, similar to that adopted to subdue many other specific and refractory inflammations which we can cure only by substituting, by means of therapeutic agents, an artificial phlogosis with the nature and consequences of which we are acquainted.

I have still a word to say, Gentlemen, in continuation of my comparison of pulmonary with urethral blennorrhagia. When the latter is accompanied by violent inflammation which is propagated to the bulb, when there is chordee with *ardor urinæ*, the balsams, acting more energetically than was expected, may exasperate the irritation of the affected parts, and prove more injurious than useful. In the same way, when in bronchial catarrh, the inflammation by extending to the pulmonary parenchyma, lights up a general febrile condition, balsamic medicines are contra-indicated. Before we employ them in such cases, we must allow the inflammatory fever to subside; otherwise, we shall bring on complications of a character more serious than those which we desire to subdue.

Besides the treatment which I have now been recommending, there is another method which you have seen me employ concurrently with it in bronchial catarrh. It is likewise topical, but it is more direct in its action than the treatment of which I have already spoken. I refer to the *inhalation of medicinal substances*, which bears the same therapeutic relation to pulmonary blennorrhagia which stimulating injections bear to blennorrhagia of the genito-urinary organs. These medicinal inhalations admit of being very much varied, both in respect of the substances employed and the manner of employing them.

The simplest mode of administration consists in causing the patient to inhale air impregnated with balsamic vapours. To accomplish this object, you place in the patient's room vessels containing tar, on which, morning and evening, you pour a small quantity of essential oil of turpentine, mixing it at the same time with the tar. By adopting this plan, the patient is kept constantly in a balsamic atmosphere; and to such an extent does absorption take place, that the urine acquires a violet odour. Inhalers have been invented to contain hot water, to which are added from fifteen to thirty grammes [about from 15 to 30 fl. drachms] of tincture of

benjamin, and a little turpentine.[1] The most effectual method of bringing modifying medicaments into contact with the bronchial mucous membrane is to employ the spray-apparatus invented by Dr. Sales-Girons, an instrument which you see in constant use in our wards, and by the aid of which, I believe great service may be rendered in different affections of the respiratory passages. By using a fumigator or spray-apparatus you may obtain good results vary your remedies.

Some benefit may be derived from *mercurial fumigation*, accomplished bythe patient inhaling the fumes of metallic mercury produced by throwing mercury on a hot brick; but this proceeding has the drawback of frequently causing salivation.

Finally, Gentlemen, in the treatment of pulmonary blennorrhagia, great relief is obtained by smoking cigarettes of arseniated or nitrated paper. When I come to speak of asthma, I shall give you the formula by which to prepare them.

The different methods of treatment which I have described, will enable you beneficially to modify the character of certain catarrhal affections accompanied by muco-purulent secretion, which, when neglected, quickly become chronic, lead to dilatation of the bronchial tubes and vesicular emphysema, ending by being, if not diseases, at least serious infirmities.

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[1] Gaujot :—Arsenal de la Chirurgie Contemporaine. Paris, 1867. T. I, p. 121.

LECTURE XXIX.

HEMOPTYSIS.

Hemoptysis.—Supplementary Hemoptysis.—The Differential Diagnosis between the Hemoptysis symptomatic of Pulmonary Phthisis and the Hemoptysis of Hæmorrhagic Pneumonia is by no means so easy as some physicians allege.

Gentlemen :—A short time ago, a girl of thirteen years of age, who occupied bed 32 of St. Bernard's ward, died suddenly, death being the consequence of a terrible attack of hemoptysis, which occurred under circumstances which I shall now relate.

The patient was admitted to the clinical wards with pneumonia undergoing resolution. Convalescence, however, was not thoroughly established. The continuance of the local thoracic signs heard on auscultation throughout a great extent of lung, particularly at the summit, and the persistence of the general symptoms of the fever of tubercular consumption, clearly testified that there was very far advanced phthisis. The disease, however, did not appear to be making rapid progress. For some days, the child had been in better spirits than she had shown since she came into the hospital, when, one evening about six o'clock, nearly two hours after her evening meal, she was seized with a fit of coughing, and at the same time copious hæmorrhage supervened which led to death in less than five minutes. The patient, who retained consciousness to the last, stated that she felt herself to be dying. The blood, which issued copiously from the nose as well as from the mouth, was not frothy, and had a dark red, or almost black colour : when it had coagulated in the vessel, it was black. The hæmorrhage had more the appearance of hematemesis than of hemoptysis.

From the suddenness of the attack, the antecedents of the patient, and the rarity of hæmorrhage from the stomach at so early an age, I concluded that the bleeding was from the lungs. In coming to

this conclusion, I was also influenced by the recollection of similar cases, and in particular of one which occurred in the previous year in the same ward, the subject of which was a girl of the same age, and who likewise was suddenly carried off by a terrible attack of hemoptysis.

At the autopsy, some might at first have supposed that I was wrong in my diagnosis, and that the young girl had died of hæmorrhage from the stomach. In point of fact, the stomach was distended with blood similar in appearance to that which had been ejected during life: but we could find no lesion in the stomach. It soon became evident that the hæmorrhage was bronchial. The lungs were riddled with softened tubercles, and in the upper part of both, there were extensive cavities: from both, when cut into, a large quantity of blood welled out by the ramifications of the bronchial tubes. No ruptured vessel was found; and, strange to say, the cavities did not contain blood. The hæmorrhage was, however, not the less indubitably of pulmonary origin: blood was found in the stomach simply because the hæmorrhage being very great, had not sufficient way of exit by the mouth and nose, and the blood was consequently of necessity forced down the œsophagus.

Gentlemen, this case, and others which I have seen in the clinical wards, have made me desirous of entering with you into some details relative to the diagnostic and prognostic value of hemoptysis. The first idea suggested by seeing a patient spit blood is, that he has tubercles in the lungs. Without thinking of his age, or the special circumstances in which he is placed, we are apt to jump to the conclusion that he has tubercles, and is the subject of a threatening phthisis. Nevertheless, if I were to reckon up all the cases of pulmonary hæmorrhage which I have met with in hospital and private practice, I believe that I should find, that in the majority of cases, the bleeding did not depend on tuberculosis. However paradoxical this opinion may seem to some physicians, it is not the less a truthful statement.

There is a certain class of cases of hemoptysis which is seldom met with in hospitals—cases in which the hemoptysis is the result of *hæmorrhagic deviation*. We meet with women, subject to nervous attacks, who spit blood, sometimes in considerable quantity, though they do not experience any marked disorder of the menstrual function. Attentive examination of the thoracic organs reveals no lesion of the respiratory or circulatory apparatus. Neither do the

patients present any symptoms of pulmonary or cardiac disease. When they reach the change of life, the hemoptysis ceases, never to return.

There are also some women, who during pregnancy, and others, who during the whole time they are nursing, spit blood: the hæmorrhage ceases spontaneously after delivery, or at the end of lactation, as the case may be; and is not symptomatic of pulmonary tubercle nor of cardiac disease.

You have had an opportunity of seeing a case of this kind. The patient was a nursing woman who came into our wards, after having had attacks of profuse hemoptysis at about the tenth month of an engagement as wet-nurse at Paris. These attacks recurred at very short intervals: the secretion of milk was dried up: the patient fell into an anæmic state: and I could not get rid of the idea of tuberculosis, although auscultation and percussion revealed no positive signs of such an affection. This woman left the hospital, to return to her native place. Two years afterwards, Dr. Blondeau had an opportunity of seeing her. She had then been for a long time in good health, and had regained a plump appearance, as well as a good colour: she had recently given birth to a very healthy infant; and was again in a situation as wet-nurse.

How are we to explain these cases of hemoptysis? I cannot answer that question; but I have now become sufficiently instructed in the subject by experience to be less alarmed than I used to be by hemoptysis supervening in the circumstances we have now been considering. It is an interesting fact in relation to this class of patients, that they are generally nervous, and sometimes also subject to menorrhagia, at least to a very abundant menstrual flow. They seem to be under the influence of a hæmorrhagic diathesis, and when the normal crisis does not take place from the mucous membrane of the uterus, it takes place from the mucous membrane of the bronchial tubes. Although these bronchial hæmorrhages are not such formidable occurrences as one might be inclined to believe —although they may recur, at more or less regular intervals, during many years without occasioning danger—it must not be forgotten that their frequent recurrence causes congestion, which may give rise to inflammation of more or less dangerous character, and provoke diasthetic manifestations which, were it not for the exciting cause, would not probably have been produced.

When commencing practice, I used to be frequently called to a

lady who had suckled four children, and had had violent attacks of hemoptysis during the lactations. For some years, menstruation had been exceedingly profuse, a circumstance which made me uneasy about her. A long period elapsed before I could make out anything abnormal in the state of the uterus; nevertheless, this patient died of uterine cancer. I may add, that she was rheumatic, and subject to serious nervous symptoms.

As an example of hemoptysis coincident with a kind of hæmorrhagic diathesis, I shall now relate a case. Among my old friends, there is a lady, who is the mother of an eminent physician. During childhood, she had had fits of somnambulism; and ever afterwards, she was subject to nervous symptoms of the most curious description. At present, she still experiences, upon the slightest emotion, partial congestion of the skin, as is seen by its assuming a scarlet colour lasting some minutes. Up to the time when the catamenia ceased, she was subject to menorrhagic attacks, which were often very alarming. When about thirty years of age, she had had such profuse hemoptysis accompanied by so great an amount of dyspnœa, that my accomplished friend Professor Andral, though unable to detect any signs of phthisis, judged it right to send her to the Eaux Bonnes. This lady now has emphysema of the lungs. Age has deadened the nervous excitability, which in her earlier life was manifested by the phenomena which I have described, and although her health is far from being so good as could be desired she still looks fresh and plump. Neither she nor her children have any symptoms to lead to the belief that they have tubercles.

Hemoptysis besides proceeding from the peculiar condition or diathesis of pregnant and nursing women, may be to a certain extent a physiological occurrence, if such a term be applicable to a hæmorrhage taking the place of a natural or accidental sanguineous discharge, which from some cause or other has been prevented from finding an exit by the usual outlets. Thus it is, that in women in whom menstruation is irregular or suppressed, hemoptysis is one of the most common forms in which hæmorrhage occurs as a supplement of the menstrual flux.

When in addition to the now described peculiar tendency in the economy, there is a local predisposing cause, it is obvious that the pulmonary hæmorrhages will occur still more easily. We can understand that such will be the case in women having pulmonary tubercles, heteromorphous products, here playing the part of

Van Helmont's thorn, and occasioning a state of congestion of which bronchial hemorrhage is the consequence.

We have seen an example of this in a young woman, who occupied bed 25 *bis* of St. Bernard's ward. This patient had been recently delivered, when she came into the Hôtel-Dieu. She then was nursing an infant, very soon afterwards carried off by pulmonary phthisis, of which the mother herself presented the symptoms and the signs. She had frequent cough, muco-puriform expectoration, fever, night-sweating, dyspepsia, and considerable loss of flesh. She had had antecedent hemoptysis. The physical examination of the chest yielded, on percussion, a harsh sound at the right apex, both before and behind: on auscultation, there was heard, in the same region, prolonged expiration, moist crackling, and coarse mucous râles. These phenomena became modified: the patient regained a certain amount of plumpness, and her strength returned: where the local signs had been so well marked, there was now heard only feeble respiration, without any râles: the only remaining symptom was dyspepsia indicated by a feeling of weight at the stomach after meals. This dyspepsia yielded to the administration of three drops of hydrochloric acid, which was taken daily in sugared water immediately after breakfast. I was hoping for, and even announcing, a speedy cure, when, on the 18th May, the patient was seized with hemoptysis. She ejected blood from the mouth, which came up as if by vomiting: in the spittoon one could distinguish sanguineous sputa, frothy sputa of vermilion red, viscid, dark red, and black sputa exactly like that which is characteristic of pulmonary apoplexy. For four or five days, there was a recurrence of the hemoptysis, which came on in the evening or during the night. It yielded, or at least it seemed to yield, to the use of terebinthinate draughts, decoction of rhatany, and *eau de Rabel*.[1] The patient, however, exhausted, and also alarmed by the loss of blood, again fell off in strength and plumpness. Nevertheless, she was beginning to recruit, when, after the lapse of a month, on the 18th June, there was a recurrence of

[1] Eau de Rabel is a mixture of sulphuric acid and alcohol, which takes its name from Rabel the person by whom its virtues were first extolled. It consists of one part of sulphuric acid to three of alcohol. The acid is added little by little to the alcohol; and after eight days the mixture is decanted. It is given internally as a stimulant, tonic and astringent in doses of from 10 to 100 drops in mucilage. Pure, it is a powerful topical styptic.—Translator.

the hæmorrhage, which returned repeatedly during two days. Having learned on this occasion that she had not menstruated since her accouchment, it occurred to me that the hemoptysis was periodical, and depended upon a hæmorrhagic deviation. The first application of a leech to the inside of each knee prevented the return of the hemoptysis; but the sputa continued to be sanguinolent, and in colour were like the lees of wine. The small local derivative bleeding was repeated on the 22nd and 24th June, after which latter date, there was no more sanguinolent spitting.

You have observed that from that date, I watched the symptoms of uterine congestion. Every twenty or twenty-two days, this woman had slight headache, a feeling of weight in the lumbar region, pains in the hypogastrium, and a more frequent desire to make water. You then saw me apply on three consecutive days a single leech to the inside of one of the knees. By this proceeding, I have been able to prevent return of the hemoptysis, and have seen a retrocession, or at least an absence of aggravation, in the pulmonary symptoms. The patient left the hospital, carrying with her the cause of death inevitable and probably near; but she left the hospital in infinitely better health than that in which she entered it.

The differential diagnosis in this case, though difficult, on account of the pathological elements being so commingled, appears to have been justified by the mensual periodicity of the symptoms, and the success of the treatment employed.

Professor Andral says that periodical hemoptysis in women having tuberculosis ought not to be regarded as a supplementary hemorrhage, that it is associated with the existence of tubercle, and that its return no doubt depends on a more acute congestion taking place *each month* in the lungs, around the tuberculous masses.[1]

This remark of Professor Andral does not appear to me to weaken the view which I have taken; for it remains to be asked, whether this more acute congestion which takes place *each month* ought not to be regarded as an accidental physiological action set up by the presence of the heteromorphous products in the lungs, which play the part, as I have already said, of Van Helmont's thorn; but which is dependent likewise on peculiar conditions which escape us,

[1] Andral:—in a note at p. 307 of the 1st volume of Laennec's "*Traité de l'Auscultation Médiate.*"

and under the influence of which, irrespective of any tubercular affection, hemoptysis is produced, as a supplement to menstruation in women whose courses are irregular. Cases of this kind are not very common, but they do unquestionably sometimes occur.

Be that as it may, one can see, that in similar circumstances, the prognosis of hemoptysis has a degree of seriousness very different from that of which I spoke when considering the hemorrhagic deviations which occur without local exciting causes. Here indeed, the symptoms are complicated by the local lesion which has produced them, just as the lesion itself is necessarily complicated by the existence of inflammatory congestive hæmorrhage which at each return, must accelerate the evolution of hæmorrhage.

Supplementary hæmorrhages are rare in hospital practice; but hemoptysis symptomatic of tubercular phthisis is not perhaps the hemoptysis most frequently met with: the most common kind is that dependent on diseases of the heart.

This remark must not be taken to imply that tubercular hemoptysis is absolutely less frequent than hemoptysis arising from cardiac disease. I merely mean to say that in tubercular subjects, the attacks of hemoptysis are transient, and occur at the beginning of phthisical disease, at a stage when the patients do not come into hospital. Hemoptysis, on the contrary, dependent on cardiac lesion, occurs when disease is far advanced, and consequently at a time when the sufferers are obliged to seek relief in hospitals.

Let us pause for a few minutes, to see if we can place upon a proper footing the differential diagnosis of the two kinds of pulmonary hæmorrhage of which I have been speaking.

During youth, adolescence, and the first epoch of mature age—from the age of 16 to 40—hemoptysis is generally dependent on pulmonary tubercles. When met with during these periods of life, whether in hospital or private practice, we may say in the words of the aphorism of Hippocrates—"*ab hæmoptoe tabes.*" But after forty, and still more after fifty, it is, as a general rule, the sign of disease of the heart, and not of tubercular phthisis: at that period of life, even when the sputa have not that sanguinolent character attributed to apoplectic sputa, when they are of a vermilion colour, frothy, or somewhat fluid, auscultation will disclose the signs of cardiac lesion. But in youth and mature age, though the sputa present the characteristics supposed to belong to the sputa of pulmonary apoplexy, though they are black, viscid, and unmingled with

air (as is not at all unusual and as sometimes occurred in the young woman with phthisis, whose history I have just related), the probability is that the hemoptysis is symptomatic of the presence of tubercles, and that, sooner or later, auscultation of the chest will give positive confirmation of that diagnosis.

There are, of course, exceptions to these rules. Even in very young subjects, hemoptysis may be the consequence of disease of the heart, just as in old people it may be symptomatic of pulmonary tuberculisation. These exceptions, however, do not weaken the general rule which I have stated.

In pulmonary phthisis, sanguinolent expectoration supervenes either prior to every other manifestation of the disease, or after there is undoubted evidence of its existence.

Laennec regarded the hemoptysis of pulmonary phthisis as not profuse, as frothy, and as sometimes clotted, particularly towards the end of the attack. According to him, the very copious hemoptysis, in popular phraseology designated "vomiting of blood," is almost always due to pulmonary apoplexy. Professor Andral is opposed to this view, maintaining that the illustrious inventor of mediate auscultation had observed far fewer patients in his private than in his hospital practice, where, as I have said, tubercular hemoptysis is rarely met with. No doubt the quantity of blood lost in these cases is generally small, but there are terrible cases in which death is caused by the enormous extent of the hæmorrhage. I have seen three cases of this kind; and the subject of one of them was one of the young girls to whom I referred at the commencement of this lecture. In her, the blood was frothy, and bright-red [*rutilant*] when its flow was not profuse: black and clotted, when it was poured into the bronchial tubes too rapidly to be mixed up with the blood.

In diseases of the heart, the consecutive hemoptysis is still less violent than bronchial hæmorrhage of tubercular origin. We see cardiac cases in which the bleeding recurs on fifteen, thirty, or even fifty consecutive days without causing death. Of course, if it depend on the rupture of an aneurism into the bronchial tubes, it proves more rapidly mortal than phthisical hemoptysis.

I have already said enough to show you that the age of the subject, and the manner in which the symptoms advance, are important elements in the differential diagnosis which I am now endeavouring to establish between the two different kinds of hemoptysis. It is a noteworthy point in relation to the seat of the hæmorrhage, that in

phthisis the blood generally comes from the bronchial surface, and in heart affections most frequently from the parenchyma, taking place, in the first instance, into the vesicles of the lungs.

Let us now study the distinctive characters of bronchial and pulmonary hæmorrhage; and inquire whether their distinctive characteristics are as accurately defined as some maintain.

Bronchial hæmorrhage, it is said, occurs in the form of sanguinolent sputa, frothy, to a certain extent diffluent, and in fact presenting the appearance of whipped air and blood, or of the froth produced in a vessel into which an animal has been bled: it has a bright-red hue [*une rutilance*], which, to a certain extent, is regarded the classical sign of this kind of hemoptysis. Again, it has been said that sometimes the blood flows profusely—an opinion opposed to that of Laennec, as we have seen—and at other times, in small quantity; that is to say, that sometimes the subjects of these accidents will for several days expectorate matter tinged with bright blood, while at other times, they will seem to *vomit* a quantity of blood sufficiently large to abruptly terminate life. Finally, it has been said that in these hemoptoic expectorations, there is no admixture of food or mucus.

It is much to be regretted that the characters are not always so well defined as now represented in description. Of this you will see a striking example, in a woman occupying bed 27, St. Bernard's ward. She is a phthisical subject without any lesion of the heart, and has hemoptysis constituted of ropy sanguinolent sputa, such as are seen in the first stage of pneumonia, or in pulmonary apoplexy. This probably arises in her case and similar cases, from there being, in addition to the hæmorrhagic affection, a slight inflammatory action, which imparts to the sputa the viscidity characteristic of peripneumonia. Or, it may arise from the hæmorrhage having been rather abundant, and the lung being at the same time, sufficiently tolerant of the presence of the blood, to allow it to accumulate and remain for a certain time in the pulmonary vesicles. Under these circumstances, should no new hæmorrhage occur, the patient will, after a few days, bring up black sputa, and they will sometimes be as black as the sputa of pulmonary apoplexy. This colour is explained by the sputa not having been in contact with air, which, when it mingles with the blood, renders it red and frothy.

In cases in which the hemoptysis is unquestionably connected with tubercular phthisis, we find the expectoration mingled with portions of

food, as in the case of the patient whose history I have given you. In that case, the spittoon contained sanguinolent diffluent sputa, mixed up with a considerable quantity of vomited food and mucus.

The stethoscopic signs of bronchial hæmorrhage are often at fault. Nothing more perhaps than mucous râles will be detected upon the most careful auscultation of a person who has been spitting blood for a long period. At other times, we may hear subcrepitant or moist râles, attributable to blood in the bronchial tubes, but which as they are also heard, when there is no hemoptysis, in the first and second stages of tubercle, are not of diagnostic value. To be of real diagnostic value, it is essential that the râles should not have been heard prior to the occurrence of the sanguinolent expectoration, and that when it ceases, they too should cease. It is evident, therefore, that there are absolutely no stethoscopic signs of hemoptysis. The stethoscopic signs which may belong to it, belong equally, and indeed perhaps more, to the pulmonary lesion upon which it depends.

Generally speaking, upon opening the bodies of persons who have died after having had attacks of bronchial hæmorrhage, we find nothing more than the morbid appearances of phthisis, and a redness of the bronchial mucous membrane, due probably to imbibition. If cavities exist, they may contain a certain quantity of coagulated blood, particularly if the vascular ruptures have taken place in large cavities: under other circumstances, there will be found little blood accumulated in the bronchial tubes.

Before proceeding to the comparative examination of the sputa of *pulmonary hæmorrhage*, I have a word to say regarding this affection, with a view to point out that it is a mistake, in my opinion, to employ *pulmonary apoplexy* as its synonyme.

Pulmonary hæmorrhage generally supervenes during the progress of heart disease. On making the autopsy of individuals who have had this kind of hæmorrhage, we generally find small portions of lung which are centres of congestion, as dark in colour as the spleen, and as hard as pneumonic nuclei in their second stage. The lung tears under the fingers, and presents the granular appearance of hepatised tissue, with this difference, as Laennec remarked, that in inflammatory hepatisation, the vermilion colour of the inflamed pulmonary tissue enables us to distinguish the black pulmonary spots, the vessels, and the slight partitions of cellular tissue which separate

the lobules of the lung: but in hemoptoic engorgement, the indurated part presents a perfectly homogeneous colour, almost black, or very deep brown red, which renders it impossible to recognise in the pulmonary texture more than the bronchial tubes, and the largest blood-vessels, the tunics of which have lost their white appearance from being soaked in, and stained with, blood. During last month, you had an opportunity of seeing these anatomical characters at the autopsies of two of our patients who died from heart disease. In these cases, the lesions were made known during life by the signs of that morbid state to which the name of *pulmonary apoplexy* has been given, an objectionable designation for which *sanguineous infiltration* ought to be substituted. The affection has in fact no characters in common with cerebral apoplexy with which some wish to compare it: the term *apoplexy* always implies the idea of sudden seizure and active congestion, characters belonging much more to bronchial than to pulmonary hæmorrhage, which latter is usually more or less passive. It is true that cases have been recorded of true pulmonary apoplexy occasioning sudden death, and presenting at the autopsy more or less extensive effusion of blood into the middle of a lacerated lung, presenting very nearly the same appearance as cerebral tissue into which there has been violent hæmorrhage. *Apoplexy* is a term which would be much more applicable to active congestion of the lung, a disease which is not very uncommon, but which is very seldom accompanied by sanguineous effusion which can be properly called a hæmorrhage. Dr. Gendrin[1] has substituted for pulmonary apoplexy, the term *"pneumo-hémorrhagie,"* which succinctly expresses, without any ambiguity, *extravasation of blood into the tissue of the lungs.* He rejects the term "apoplexy," because the invasion of the disease is seldom sudden, and is not accompanied by rapidly dangerous symptoms like those of cerebral apoplectic seizures—because the alterations of tissue differ in many respects from the alterations of tissue produced by encephalic hæmorrhage—and because, in a word, it does not embrace all the forms and degrees of the pathological state in question.

To return to the subject more immediately before us, let me ask: —What are the characters of hemoptysis in cases of sanguineous pulmonary infiltration?

We are told that the sputa are sanguinolent, copious, mixed with

[1] GENDRIN:—Traité de Médecine Pratique. T. i, p. 638.

air and viscid like the sputa of peripneumonia, excepting that they are not frothy. This description may be considered as generally applicable. The sputa of parenchymatous pulmonary hæmorrhage certainly are viscid and aërated, but have sometimes a bright-red colour [*coloration rutilante*] like the sputa of the patient who occupied bed 17 of St. Agnes's ward, who, after having had attacks of pulmonary hæmorrhage, sank under disease of the heart: sometimes, they are blackish, or very deep red, a colour which, as I have already remarked, is met with in certain cases of tubercular bronchial hemoptysis.

The sanguinolent expectoration of parenchymatous hæmorrhage may assume the appearance of bronchial hæmorrhage in so far as to become frothy, a character dependent upon the quantity of blood which is brought up. Indeed, in contradiction of what has generally been said, it may be stated, that if the blood escape in small quantity, if brought up after having been slowly infiltrated into the pulmonary parenchyma it is not frothy, because it is not mixed with air. But if the hæmorrhage take place suddenly, if the blood is thrown off pretty copiously, if it flow briskly from the bronchial tubes, it will be whipped up with the air therein contained, and in this way, the expectoration will become frothy.

In the man of whom I have just been speaking, the hemoptysis presented this double character. There was some bright-red frothy sputa (exactly similar to the hemoptoic expectoration seen in phthisis) mixed with other sputa, which were viscid and of a darker colour, while some were quite black. We shall find, on making the autopsy of this patient, that the opinion formed during life was correct, to the effect that his lungs are without trace of tubercle.

In these cases of pulmonary hæmorrhage, from the stethoscopic signs being so uncertain, and the diagnostic difficulties so great, Professor Bouillaud has said, that the nature of the disease has to be divined rather than diagnosed.

Should the sanguineous infiltration have been extensive, should large hæmorrhagic nuclei exist, there will be heard, around the points invaded by the hæmorrhage, local signs similar to those of pneumonia—a blowing sound, subcrepitant, and sometimes crepitant râles. Should the nuclei be circumscribed and disseminated, in place of being somewhat extensive, the blowing sound will be absent, and the râles only will be heard. These râles are caused by the exudation of blood around the hæmorrhagic nuclei, and into the neighbouring minute divisions of the bronchial tubes. Like the

mucous râles, they are produced by the passage of air through a liquid. These signs, which be it observed belong equally to congestion of the lung, to engorgement, or to catarrh of the small tubes, may be entirely absent: if the hæmorrhagic nuclei are not only small, but situated at a distance from the surface of the lung, the most that we shall be able to hear will be coarse mucous râles in the large tubes.

Cardiac lesions are very frequent causes of pulmonary hemorrhage: the cardiac lesion which is the most common of these frequent causes is contraction with inadequacy of the mitral valve. The hæmorrhage will be the more apt to occur, if, along with the lesion of the auriculo-ventricular orifice, there is, as is usually the case, hypertrophy of the ventricles.

These hæmorrhages are in some cases very considerable, and recur three, four, six, eight, or ten times in the course of the disease of the heart; in other cases, not often, however, they are insignificant in quantity, very transient, or altogether absent. When the cardiac affection is far advanced, the patients may go on spitting blood for a month, or even up to their death.

I was lately seeing, at the hôtel des Princes, an American gentleman of sixty-five, who, consecutively to repeated attacks of articular rheumatism, became the subject of chronic endocarditis, with contraction of the auriculo-ventricular opening, and insufficiency of the mitral valve. He had had many attacks of hemoptysis which did not continue more than a few days. Six weeks before his death, these attacks recommenced, and to the last, the patient brought up by the mouth every day four or five tablespoonfuls of blood. In this patient, at first, the signs furnished by auscultation of the lungs were negative: afterwards, we heard subcrepitant râles, and a slight blowing sound. These signs did not appear till near the close of life. The blowing sound was heard throughout the whole of the right lung.

At the time I was attending the American gentleman, I was seeing along with another physician, a gentleman of sixty-four years of age, who had formerly come to me in my consulting-room. At the end of last autumn, he had been suddenly seized, after a hunting party, with difficulty of breathing and very acute pain in the region of the heart. The malady was almost overlooked by the patient, who did not give himself much concern about it. But from the symptoms becoming more severe he came to consult me. I had no difficulty in recog-

nising the existence of pericarditis, for the effusion into the pericardium was such, that, approximatively, it might be estimated at half a litre [nearly 18 fl. ounces], due allowance being made for the extent of the precordial dulness and the degree of arching of the chest, as well as to the sounds of the heart being inaudible from their great distance from the ear. Under the influence of repeated bleedings, flying blisters, and the use of preparations of foxglove, the pericarditis disappeared. Some months later, I could not detect the least sign of that affection; but along with the first and second sounds of the heart, I heard over the apex, a harsh blowing sound, which told me that there was a lesion of the auriculo-ventricular valve. For some days also the patient had hemoptysis, and in some parts of the chest I heard, on auscultation, subcrepitant râles and a blowing sound. My prognosis was unfavourable. After some deceitful rallies, this individual died like the American of whom I have been speaking.

It is usual for these attacks of hemoptysis to become more frequent and more profuse, as the disease of the heart advances.

I have now spoken of the different kinds of hemoptysis, and of the difficulty which often occurs in practice of distinguishing the one from the other; but I have still a few words to say upon the differential diagnosis of hemoptysis and hematemesis.

Gentlemen, I do not think that this differential diagnosis ought ever to be very embarrassing. It appears to me, that failing the precursory symptoms, usually in themselves sufficient to inform the physician whether the blood ejected by the mouth has come from the lungs or stomach, there would still be no room for mistake, as the manner in which the blood is ejected, and the physical characters which it presents are distinctively characteristic. It is said that hemoptysis takes place after efforts to cough: the blood then coming from the lungs is at the time of its expulsion fluid, red, and frothy; on the other hand, in hematemesis, the blood ejected by vomitive efforts is often set in coagulated masses, is black, and non-aërated. Further, it is almost always mixed with alimentary substances. Finally, this vomiting of blood—this hematemesis—is frequently followed by black stools, to which we give the name of melæna.

While it is quite true that in general the differential diagnosis of hemoptysis and hematemesis offers few difficulties, there are exceptional cases in which hesitation is quite allowable.

There may be something in the physical characters of the blood,

and in the manner in which it is ejected, to oblige us to hesitate in our diagnosis. I have already called your attention to the fact, that the blood may be black in hemoptysis when it is ejected very rapidly and with force. On the other hand, persons affected with hematemesis may bring up perfectly liquid, bright-red blood. This occurs when, owing to the hæmorrhage from the stomach being copious, the blood does not remain sufficiently long in the stomach to be acted on by the gastric secretions.

We must not attach too much diagnostic value to the manner in which the blood is expelled, nor to the presence or absence of alimentary substances, because as I have already said, violent hemoptysis and vomiting take place exactly in the same way, without any preceding efforts to cough. In hemoptysis, patients frequently eject the contents of the stomach, true vomiting being excited by the efforts to expectorate, or by the titulation of the uvula causing sympathetic contractions of the stomach. On the other hand, in hematemesis, the blood may be poured out in perfect purity, unmixed with food, bile, or mucus. And this takes place, not only when the gastrorrhagia is consecutive to the rupture and perforation of a blood-vessel, but also, even when it is symptomatic of an organic affection, and not dependent on any appreciable vascular lesion.

The malænotic stools do not in themselves absolutely declare that the hæmorrhage is from the stomach. No doubt, in hematemesis, the stools are nearly always black, but then they may also be black when the blood is primarily from the lungs, as it may have passed down the œsophagus into the stomach; as occurred in the young woman of St. Bernard's ward, whose case I mentioned at the beginning of this lecture.

Again, hemoptysis supervenes pretty frequently in patients who have neither tubercular nor cardiac disease. When speaking of bronchial dilatation, I dwelt upon the fact that spitting of blood often takes place in cases in which, at the autopsy, no tubercles can be discovered. Hemoptysis also, is often observed in connection with hydatids of the lung. We have at present an example of this in a young man of seventeen years of age who occupied bed 9 of St. Agnes's ward.

I can add nothing to what I have told you a hundred times about the treatment of pneumorrhagia or parenchymatous hæmorrhage from the lung. When it is, as is usual, connected with dis-

case of the heart, decided benefit is obtained by very moderate bleedings, the preparations of digitalis in full doses, acids, and rhatany. It is specially necessary to moderate the intensity of the determination of blood to the lungs, which, when it forms nuclei near the surface of the pleura may lead to inflammation of that membrane, and become the cause of pleuritic effusion, constituting a formidable complication of disease of the heart. You saw a case of this kind in a man who came into our wards in June 1863.[1]

When the parenchymatous hæmorrhage is obstinately recurrent, ipecacuanha is a remedy which seldom fails. I am not at present referring to ipecacuanha administered as an emetic, which is more to be relied on in the treatment of what is called bronchial hæmorrhage.

You remember an old man, aged sixty-two, who lay in bed 7, St. Agnes's ward. He was resident in the hospital from the beginning of 1863; and during the preceding year, he had asked my advice on account of his having serious symptoms of tubercular disease. For several years, he had been phthisical; and from time to time, the upper lobe of the right lung, in which there were large cavities, became the seat of acute inflammation, by which life was placed in jeopardy. Twice, within the space of five months, he had frightful hemoptysis: twice it was immediately arrested by four grammes [rather more than a drachm] of powder of ipecacuhan, administered within the space of half an hour, in such a way as to cause violent vomiting. A similar result was obtained, you remember, in the young man who occupied bed 8 in the same ward; and also in another patient now occupying bed 16.

Some months ago, I was summoned in consultation to a provincial town, in the case of a tuberculous man, aged forty-two, who had had hemoptysis going on for forty days. A great diversity of very rational plans of treatment had, in succession, been fruitlessly employed. I recommended three grammes [46 grains] of ipecacuhan to be divided into four packets, one of which was to be given every ten minutes. The hemoptysis had ceased before the last vomiting took place; and from that time, when it did recur, it was only to an insignificant extent.

Should, however, there be a relapse of the hemoptysis, the use of

[1] This case will be found fully detailed in the lecture on paracentesis of the chest—Lecture XXXII.

the ipecacuhan must be resumed. I never hesitate in such circumstances to return to it two or three times, if necessary, and I have never yet seen the least inconvenience result from this proceeding. Gentlemen, this is not a new method of treatment. For the last two centuries, physicians have lauded the Brazilian root as a remedy in all forms of hæmorrhage; and Baglivi says:—"*Radix ipecacuanhæ est specificum et quasi infallibile remedium in fluxibus dysentericis, aliisque hæmorrhagiis.*"

Nevertheless, Gentlemen, the hand trembles when it administers this remedy for the first time in the treatment of hemoptysis. We are accustomed to prescribe the greatest possible quietude to our hemoptoic patients: we counsel them to keep absolute silence: we tell them to restrain the slightest effort to cough: the very most we allow them to do is to breathe, and so frightened are we for congestion, even passive congestion of the lung, that we act as if we placed them in peril by permitting them to make the slightest effort. Yet here we are giving a medicine which produces vomiting, during which the face swells, the blood stagnates in the veins by which it is being conveyed to the auricles; and consequently, the pulmonary veins become distended. One might expect that such treatment would cause the hemoptysis to return in a much more profuse degree; but in place of this, it is stopped in nearly every case. Here is one proof more of the small reliance to be placed on theoretical explanations, and of the value of empirical facts, without which, indeed, therapeutics would be a nullity.

LECTURE XXX.

PULMONARY PHTHISIS.

RAPID PHTHISIS.—ACUTE PHTHISIS, OR GALLOPING CONSUMPTION.—*Rapid Phthisis is simply Ordinary Phthisis accomplishing its course in a very Short Period of Time.—Acute Phthisis is a Distinct Morbid Species, of which there are Two Forms, the Catarrhal and the Typhoid.*

GENTLEMEN:—You have seen in bed 5, St. Bernard's ward, a young woman between twenty-four and twenty-five years of age, the subject of *rapid* phthisis. Take special note that I do not use the term *galloping* phthisis. I purposely avoid employing that word. This does not arise from my having any repugnance to a universally accepted epithet, but because the epithet with a great many physicians has a meaning totally different from *rapid*. This, therefore, is a point upon which you are entitled to an explanation. Before I give it, however, let me succinctly recapitulate the history of our patient.

This young woman was confined on the 14th March. When upon several occasions, I interrogated her regarding her antecedents, with a view to discover whether she had any previous symptoms of chest disease, she replied, that no one was less subject to catarrhal affections of the chest than she was. She said that from time to time, she had colds in the head, but had never had cough.

She became pregnant eleven months ago, and during the whole of her pregnancy, she had remarkably good health. Labour was easy, and in all respects propitious. Some days after delivery, that is to say, on the 23rd of last March—five weeks ago—she began to cough. From the first, her cough was severe, though it could not be called very violent. Not being able by any means to get rid of it, she resolved to come into the hospital.

At my first visit after her admission, I detected by percussion a notable diminution of sound on the right side of the chest, posteriorly, in the infra-spinous scapular fossa, particularly between the scapula and the vertebral column. We also heard, by auscultation, in the same situation prolonged expiratory sound, almost bronchial blowing, mixed with moist râles. On the left side, the respiratory murmur and the thoracic resonance presented nothing abnormal. The patient had fever; but no night sweating, no morbid affection of the digestive canal, and no tendency to diarrhœa: on the contrary, she had constipation.

What was the nature of this woman's disease? She had been ill for a fortnight. The signs furnished by auscultation and percussion led me to conclude that there was induration of the pulmonary parenchyma; but the obscurity of the sound in the infra-spinous scapular fossa, the moist râles, the prolonged expiration, and even the expectoration, which had the characters of phthisical expectoration, the globular, nummular sputa floating in nearly clear serosity—these signs were insufficient to convince me that the malady was tubercular. Such a conclusion seemed all the more unsound from the patient stating that a fortnight ago, she was in the enjoyment of perfect health, and that she had never had the slightest attack of chest disease. I was therefore inclined to believe that there was pneumonia of the summit of the right lung, although the elements of a correct diagnosis were so incomplete as to cause me to have some doubt on the subject.

However, by attentive daily auscultation, I found that the blowing, in place of decreasing, increased notably every day. The mucous râles became changed into crackling. Eight days after the arrival of this woman in our wards, I began to hear, on the left side, a little of the prolonged expiration, and some subcrepitant râles: conditions similar to those on the left side were then recognised on the right side: expiration became more and more blowing: the râles became converted into crackling: and at last, we heard gurgling on both sides.

Thus, an opportunity was afforded of being present to witness the advance of the disease: we saw the seizure take place in the hitherto healthy side, not as in pneumonia, but as in tuberculisation. Hesitation as to the diagnosis was no longer possible. It was only too evident that there was tubercular induration of the summits of both lungs, that phthisis progressing with fearful rapidity was

threatening to carry off this young woman in a very brief space of time—perhaps in two months, in six weeks, or even sooner.

That, Gentlemen, is an example of *rapid phthisis*. Another example was lately presented to our notice in the youth who lay in bed 2, St. Agnes's ward.

The young man to whom I refer, who came into hospital on the 30th January, had no chest symptoms till ten days prior to that date: he died on the 25th March. A month before his death, and thirty-five days from the beginning of his illness—that is to say, on the 25th February—I detected hydro-pneumo-thorax, one of the most serious complications of pulmonary phthisis. At the autopsy, we found three perforations in the anterior and lateral part of the right lung, and in both lungs a vast number of tubercular masses of the size of a pea: there were no cavities.

With the exception of the rapidity of its pace, this form of the disease, to which we apply the term rapid, presents the same symptoms during life, and the same anatomical lesions after death as ordinary phthisis, the progress of which is generally chronic. It is the same disease as ordinary phthisis, though it generally runs its course with much more rapidity. There are also cases to which the term *latent phthisis* is given, because the symptoms remain obscure, and are masked by complications which are apt to lead us astray in our diagnosis. Nevertheless, whether the form be rapid or latent, regular or irregular, it is, I repeat, always the same disease. But *acute*, or *galloping phthisis* as it is more generally called, is not the same disease as ordinary phthisis.

The anatomical character of galloping phthisis is the presence in the entire thickness and in every part of the lungs, from base to apex, of yellowish grey, semitransparent granulations. This specific character, recognised by men of high authority, by Rokitanski among others, is not denied by any one in the present day; but there is a great diversity of opinion as to the nature of these granulations.

According to some pathological anatomists, *granulations* differ in no respect from tubercles, of which they present the ordinary microscopical characters—they are "globules," to use Leudet's words, "round or ovoid, and angular in their outline, containing a more or less transparent matter, and molecular granulations; and there is also, particularly in the semitransparent greyish tubercle, an interglobular substance of a greyish yellow colour and tolerably

firm consistence."[1] These authors, then, believe with Laennec that miliary granulations are tubercles in a less advanced state of development, and while they fully recognise the fact that galloping pulmonary tuberculisation specially presents this form of semi-transparent, greyish, or nearly yellowish granulations, they likewise admit that it is altogether exceptional not to find, in addition to these granulations, traces of tubercle in a more advanced state of development, even cavernous ulceration. Finally, according to the same pathological anatomists, miliary granulations may exist in organs—in the bronchial and mesenteric glands, in the spleen, kidneys, and meninges of the brain—precisely as the yellowish tubercles of ordinary phthisis.

But opposed to these pathological anatomists, others, whose opinions possess unquestionable value, maintain that miliary granulations are morbid products quite different from tubercle. On this point, Gentlemen, let me quote the views of a man, with whose high standing you are all acquainted.

My accomplished colleague Dr. Charles Robin in a manuscript note, kindly communicated to me in relation to some pathological specimens taken from the body of a patient who died in our wards, says, that under the name of miliary tubercle, *four species* of morbid products have been described.

The *first* species, he says, consists of *concrete pus*. This was the species of miliary tubercle found in the patient to whom I alluded.

The *second* is formed by *epidermic products of the lung*. These products are most frequently met with in children, particularly in infants at the breast; but they are also found in adults. Sometimes, they are scattered here and there throughout the pulmonary parenchyma, and at other times, they exist in close contiguity to one another, being almost confluent: their starting point is the pulmonary epithelium, just as in the parenchyma of glands, different affections have, as their characteristic lesion, augmentation in the quantity and volume of these organs. These epidermic products are the least common of the four species of morbid products now under review.

A *third* species embraces the *grey or semitransparent granulations*, isolated or confluent, in the latter case constituting what is called grey infiltration. These grey granulations have a structure essentially

[1] Leudet:—Recherches sur la Phthisie Aiguë chez l'adulte. Paris, 1851.

distinct from that of tubercles. They occur in the form of isolated grains, deposited in layers, or in indeterminate masses: they are the same as *granulations*.

The meningeal granulations met with in inflammatory affections of the meninges are sometimes tubercles; but more frequently they are the peculiar productions now under consideration. Granulations of the pleuræ and peritoneum are of a different character. In these situations, this morbid product has been confounded with tubercle, even when examined by the microscope, by persons—observe! it is still Professor Robin who speaks—by persons under the influence of the old ideas of the *school of observation*, as it has been called, and who perhaps from neglecting to employ the reagents generally used, have regarded it as the corpuscle of tubercle, a special element, to which I shall return forthwith. It exists in a great number of inflammatory and other products, such as the vegetations which form on the surface of wounds, and on syphilitic mucous patches.

The following are the anatomical elements of this granular product:—

1st.—The small spherical corpuscles of which I have been speaking;

2nd.—A very considerable quantity of amorphous substance, granular, semi-solid, infiltrated into the pulmonary tissue, and filling the minute subdivisions of the respiratory passages;

3rd.—Fibro-plastic elements;

4th.—*Granular bodies*, to which the term *inflammatory* has been given; and

5th.—A small quantity of epithelium coming from the minute bronchial tubes.

It is a curious fact that occasionally, only occasionally, however, small masses of tubercle are found in the centre of these peculiar morbid products, situated it may be in the membranes of the brain, in the pleuræ, or in the lungs. This occurrence, I repeat, is not usual. It is chiefly observed in subjects who present large masses of grey infiltration. However small these tubercles may be, they have a yellowish colour. They are always insignificant in quantity, as compared with that of the granulations. These granulations have no similarity in disposition with those which constitute the characteristic element of ordinary phthisis; and it would be an error to say, as has been said, that the former are merely the latter in a less advanced stage. The latter never succeed the former species, gallop-

ing phthisis always proving fatal long before the tubercular deposit is abundant.

Thus, Gentlemen, you perceive that three species of morbid products are included in the improperly applied term—*miliary tubercle*, viz. concrete pus, epidermic productions, and grey granulations.

The *fourth* species described by Dr. Robin is only a variety of the third species—the grey granulations.

True tubercle is sometimes found along with the products called miliary tubercles. Dr. Robin says, that however small the morbid products may be which contain the tubercular corpuscles, they have always the yellowish white or yellowish grey colour characteristic of tubercle, and never the grey colour of the products anatomically characteristic of galloping phthisis. When the naked eye only is used, there may be a difficulty of distinguishing them from concrete pus, but with the others, there is no risk of confounding them: and by the microscope, they can be quite easily distinguished from one another.

In relation to the general disposition of these products, I must state, that from the autopsies I have had occasion to make, I have learned that when cavities are found in the lungs in galloping phthisis, they are simply small abscesses; and however large the cavities may be, they are never divided by bands or columns formed of shreds of cellular tissue. Again, it is important to know that the glands are only attacked in exceptional cases.

Putting aside the anatomical lesion, the nature of which is very open to discussion, the form of phthisis properly called galloping will be found to differ both from rapidly progressing and chronic ordinary phthisis. We shall see that the difference is still greater in respect of the symptoms than of the lesions.

Some of you no doubt remember a young woman of twenty-one years of age, who lay in bed 10, St. Bernard's ward. When she came into hospital, she had only been ill three months. Till then, the period at which she came to live in Paris, her usual health had been good. From that time, however, it was out of order: she had less inclination for food, and her strength was perceptibly failing. She continued, however, to attend to her domestic duties, till three weeks before admission to hospital, when she was obliged to take to her bed. At that time, she had diarrhœa and colic: at first, the diarrhœa occurred at considerable intervals; but it soon recurred every day,

and became profuse. At the same time, chest symptoms set in: she had cough and expectoration, but no spitting of blood: there was a great deal of fever.

When she came into our wards, I was struck with her appearance of prostration and stupor. The fever was intense: the skin was hot and dry: the pulse was quick, full, regular, and not rebounding. There was profuse diarrhœa: the stools were yellow.

Five days later, delirium supervened. There was a great deal of cough. Observing muco-purulent sputa in the spittoon, I was led to make a more particular examination of the respiratory apparatus. On auscultation, I heard posteriorly, disseminated over the whole of both lungs, coarse mucous râles, as well as sibilant râles. Anteriorly, percussion over the left clavicle produced the cracked-vessel sound [*bruit de pot fêlé*]: I found diminished thoracic resonance: I heard, moreover, coarse mucous râles, gurgling in fact, and at one point there was cavernous blowing. A few days afterwards, the patient died. At the autopsy, we found such lesions as I have just described to you.

What, then, Gentlemen, are the *symptoms of galloping phthisis?*

A young woman—I say a *young woman,* for it is chiefly women, and chiefly young women whom I have seen the victims of this malady—a hitherto healthy young woman, without appreciable cause, falls into an uncomfortable state of health, which it is not easy to describe: she is dyspeptic, and loses her appetite: her strength flags, and a more or less feverish condition shows the disorder which pervades her system. This state of discomfort and languor lasts from a fortnight to three weeks or a month. During this period, the patient continues to go about her usual avocations, complaining, however, all the while, of unaccustomed weakness, and of great incapacity to do anything requiring mental application. She has at the same time night sweats and a short dry cough: on auscultating the chest, we hear loud râles in various parts. When the symptoms have only existed for a few days, they are attributed to catarrh or slight bronchitis; and in fact, there is nothing in the aspect of the case to lead to any serious apprehensions. But the catarrh goes on, and the fever continues. On examining the chest, it is found that the râles have become more numerous and more moist: they are heard throughout the whole extent of the lungs, from base to apex, before and behind. Time passes on, and matters, in place of improving, become worse: there is an increase of fever:

there is insomnia: the cough, becoming more and more urgent, is accompanied by expectoration, which is at first mucous and then muco-purulent: the finest râles audible, the subcrepitant, are at some points mingled with prolonged expiration, and even with a blowing sound. The thoracic resonance on percussion remains normal. Respiration is embarrassed, short, and quick; and the dyspnœa increases to such an extent that the patient is obliged to keep the sitting posture. The symptoms go on increasing in severity: the strength becomes more and more exhausted: the countenance assumes an anxious expression: the discoloration of the skin is succeeded by an asphyxial hue, and in five, six, seven, or eight weeks from the beginning of the symptoms, the patient sinks in a state of emaciation analogous to that which occurs during the course of severe fevers; but in a state quite different from the emaciation which attends ordinary phthisis.

The picture of galloping phthisis which I have now rapidly sketched would be very incomplete were I to present it to you as the absolute type of the disease. It only brings before you one form, which may be called the *catarrhal form:* there is another, the *typhoid form,* which it is quite as important to be acquainted with.

In the typhoid form, though we meet with the thoracic signs and symptoms to which I have been directing your attention, it is the general condition of the patient which characterises his malady, and to closely does this general state simulate typhoid fever that the case may be mistaken for one of that disease. The symptoms complained of by the patient, and the phenomena observed by the physician, are intense headache, a stupid expression of countenance, low delirium (changing sooner or later into more or less violent delirium), and frequently *subsultus tendinum.* The countenance in place of being pale is florid; but the red is not confined to patches over the cheek-bones, as is remarked in the subjects of ordinary phthisis, particularly during the evening exacerbations of hectic fever. There is high fever, and the heat of the skin, which is not complained of by the patient, corresponds with the acceleration of the pulse. The abdomen retains its natural degree of softness and tension. Pressure made over the right iliac fossa does not produce gurgling: there is no diarrhœa: and it is important to note that there are none of the true rosy lenticular spots of dothinenteria. In the typhoid form of galloping phthisis, the invasion of the disease is generally more abrupt than in the catarrhal form, and its beginning

is marked by more or less violent rigors. The course of the disease is also more rapid; and it terminates by asphyxia or nervous seizures.

In cases in which galloping phthisis simulates typhoid fever, examination of the temperature furnishes us with a valuable means of diagnosis. It is only in exceptional cases, that the temperature is as high in acute tuberculisation as in typhoid fever: the morning and evening oscillations too, are greater; thus, in galloping phthisis, the evening temperature differs one or two degrees from the morning temperature, while in typhoid fever, there is very seldom as much as one degree of difference between the temperature of the patient in the morning and the evening.

Neither in the catarrhal nor typhoid forms of galloping phthisis, do you find, Gentlemen, the symptoms of ordinary phthisis, even when the latter runs an exceedingly rapid course. There is, however, one point at which galloping and ordinary phthisis seem to have a connecting link: both attack persons in whose families tuberculosis is hereditary: at the same time, I must add, that they also attack those in whom no hereditary taint can be discovered.

For the reasons now laid before you, one of my former excellent pupils, now my colleague in the Faculty of Medicine, as well as in the medical service of the hospitals, Dr. Empis, has come to the conclusion, that galloping phthisis ought to be considered as distinct from tuberculisation, from which he says it differs not less in respect of its lesions than of its symptoms. With the view of fully preserving this distinction, he has given the name of granular disease [*granulie*] to the affection which is characterised anatomically by the production of granulations in the parenchyma or serous membranes. According to this doctrine galloping phthisis is the thoracic form of granular disease [*la forme thoracique de la granulie*]: the cerebral form is seen in brain fever or tubercular meningitis, and the abdominal form in cases having typhoid symptoms.[1]

In galloping phthisis, the prognosis is death. Death, sooner or later, is invariably the termination. Hitherto, Gentlemen, art has unfortunately proved unable to contend against this redoubtable malady: it is still more distressing to know that we have not the power even to alleviate the condition of sufferers by whom we may be consulted.

[1] Empis, (G. S.):—De la Granulie, ou Maladie Granuleuse. Paris, 1865.

I must give you the particulars of one more sad example of this disease :—

On the 2nd February 1861, my colleague Dr. Barth and I were sent for to Les Oiseaux convent, to see a young Spanish lady, sixteen years of age. Her ordinary medical attendant, Dr. Vosseur, informed us that this young lady had a fortnight previously begun to have uncomfortable feelings, and to suffer from fever, without experiencing any local symptoms, excepting decided oppression in breathing. As the symptoms continued, Dr. Barth was sent for, eight days later: at that visit, he was struck with the lividity of the lips and face. The lividity extended to both hands. There was great oppression of the breathing, and ardent fever. Nothing abnormal could be detected by the most careful auscultation: there was neither râle nor the sound of prolonged expiration. The functions of the stomach were as well performed as it was possible to desire.

Eight days after that visit of Dr. Barth, I again met him in consultation on the case. There was then extreme frequency of pulse and respiration, and a frightful increase in the lividity of the skin. During the night, the patient had had insomnia and some raving. Throughout the whole extent of the left lung, we heard very fine subcrepitant râles: throughout the whole of the right lung, we heard coarse subcrepitant râles mingled with mucous râles: there was no expectoration. Our opinion was, that there was very little probability of the patient surviving more than three or four days.

She died on the 4th February, seventeen or eighteen days from the beginning of the attack.

Pulmonary Tuberculisation, and Chronic Peripneumonic Catarrh in Children.

Gentlemen :—Permit me now to fix your attention for a short time upon a little patient in bed 13 of the nursery attached to St. Bernard's ward. For some time past his condition has been very anxious, and the diagnosis of his malady very embarrassing.

This child is between seven and eight years of age. Since he was about three months old, he has had a severe catarrh, accompanied by fever, which has never left him since the catarrhal malady began. He has nevertheless continued to take the breast, and it is

assuredly in consequence of his appetite for nourishment that he is still alive. He was brought here a fortnight ago. He had at that time a great deal of cough, and much oppression at the chest in breathing.

On examining the chest, I found tubal blowing on the left side, extending from the infra-spinous fossa of the scapula to about the base of the lung, and resonance of, I cannot say the voice, but of the cry: the blowing and the resonance were well-marked, particularly during expiration. At intervals, there exploded under my ear cracks of submucous crepitant râle, some of which were very fine. Comparative percussion, posteriorly, of both lungs showed us that there was very evident dulness on the left side.

The child had fever. In consideration of the general symptoms and physical signs, I thought that the case was pneumonia, or rather pleuropneumonia: I believed that the lung was indurated, and that there was false membrane on its surface. But there was still a question to solve:—What was the nature of the induration? Again, was it recent, or of old date? And again, was the induration purely inflammatory, or was it associated with the presence of accidental products in the pulmonary parenchyma? Finally, had we to do with acute pneumonia, chronic pneumonia, or tubercular pneumonia? The solution of these problems was attended with more than one difficulty.

In children, particularly in infants at the breast, and during the first three years of life, the characters of pneumonia are different from those which the disease presents in adults. In the young subjects, lobular pneumonia, such as is observed in adults, is a very rare, and not a very serious affection, whereas peripneumonic catarrh, or broncho-pneumonia, at a very early age, is one of the most dangerous diseases with which we are acquainted, inasmuch as it nearly always proves fatal.

If you study catarrh, you will find out that there is no disease so uncertain in its course. It has no fixed limits of duration: it will sometimes continue for thirty-six or forty-eight hours; and at other times, it will go on, in an acute or subacute form, for two or three months. You never can tell a patient with catarrh when he will get rid of it; whereas, when the disease is pneumonia, it is more easy to give an answer. Generally speaking, in from nine to twelve days, pneumonia terminates in death, or the general symptoms improve, and convalescence begins. Do not suppose that the un-

certainty which belongs to the course of catarrh is peculiar to bronchial catarrh: what I have said applies to catarrh in general, whether it affect the mucous membrane of the bronchial tubes, intestines, or bladder, or of the genital organs in either sex.

That proposition established, you are able to understand that as catarrh is the starting point of pneumonia in children, similar difficulties of prognosis will exist as in bronchitis: like bronchitis, it will maintain indeterminate characters, and the same tendency to relapses, to which you can assign no term.

A child is attacked with a severe feverish catarrh: at the end of four or five days, on auscultation of the chest, you hear disseminated over it subcrepitant râles, and by-and-by a blowing sound: thus you arrive at the legitimate conclusion that there is pleuro-pneumonia. To subdue this malady, you have in vain had recourse to the most energetic medicines; and some days later, the râles and the blowing sound, which had disappeared, in a very short time will be again audible. You will find them in a different point from that which they previously occupied, whether that was in another part of the same lung, or in the other lung: very soon afterwards, without leaving the newly-invaded parts, they may occupy those where they were first heard. Such is the condition of the malady, the signs of which you will recognise much better by auscultation than by percussion, which will only tell you that an entire lobe, or a great part of a lobe, has been invaded.

Thus peripneumonic catarrh may, within a few days, abandon the points which it first occupied and take possession of others, last of all, however, wholly disappearing; and thus peripneumonia may come and go successively for one, two, or three months. The successive attacks are not relapses, but returns of a cured disease. It is always the same in catarrh: the long series of interrupted and resumed symptoms, which characterise it results from a similar cause.

In the pure pneumonia of adults, matters pursue an entirely different course. A lobe is attacked: the inflammation extends to the parts in the vicinity of these which were primarily and principally affected, but it does not leap from one point to another, like catarrhal peripneumonia: it remains within the limits where it circumscribed itself from the first, or it advances step by step.

There is no difficulty in understanding, that in broncho-pneumonia, the pulmonary parenchyma, under the influence of the morbid

action of which it has been the seat during different attacks, permanently retains a more or less indurated state. Hence, it is impossible to avoid admitting, that bronchitic catarrh will soon be accompanied by chronic pneumonia; also, let me add, that chronic pneumonia is a somewhat less uncommon affection in children than in adults.

In the adult, chronic pneumonia is so rare, that (as you know), its existence has long been disputed by a certain number of physicians. However, the majority of clinical observers of the present day, while they point out its great rarity, hold that about the tenth or twelfth day of an attack of pure pneumonia, the general phenomena may disappear, the local symptoms remaining. The fever subsides: the sputa regain their natural appearance: and the appetite returns. Nevertheless, dulness on percussion remains: on auscultation, there is heard bronchial blowing, crepitant râles, bronchophony to a somewhat considerable extent; or—and many examples of this have been cited—neither normal nor abnormal sound can be heard in the seat of the lesion. This state of matters may last for fifteen, twenty, thirty, forty, and even for seventy days, as you will learn from a case detailed by Dr. A. Raymond in his thesis.[1] I should also wish, in relation to this topic, to recall to your recollection the man whom we had for so long a time in bed 19 of St. Agnes's ward, who, at the date of his admission, had acute pneumonia in a very aggravated form. In this individual, for nearly two months, we noted subcrepitant râles, and a blowing sound, on the right side, in the infra-spinous fossa of the scapula: he always retained a certain degree of fever: nevertheless, when he left the hospital, his health was quite restored, his respiration had become natural, and there was no longer any abnormal sound to be heard in the chest. It is evident that in this case, the inflammatory lesion, the induration of the pulmonary tissue, had persisted for a much longer period than is generally required for its resolution. The hepatisation, doubtless, did not keep the same form which it had at the fourth or fifth day from the invasion of the disease, but nevertheless it continued to exist, and was quite independent of any tubercular affection.

Chronic pneumonia has been correctly stated to be connected, not always (as those who deny its separate existence maintain), but

[1] Raymond, (A.):—Sur la Pneumonie Chronique. *Thèse de Paris*, 1842.

almost always, with the presence of accidental products in the pulmonary parenchyma: or in other words, this form of pneumonia is almost always tubercular. This is true in respect of adults: it is also true in respect of children; but in the latter, it is, speaking comparatively, a little more usual to meet with simple chronic pneumonia, that which terminates sometimes, though seldom, in resolution, but which under certain circumstances causes suppuration of the lobules, giving rise to small disseminated abscesses emptying themselves into the bronchi, their most propitious termination, or opening into the pleura, so causing very formidable symptoms. It sometimes happens that these abscesses become encysted in the midst of lobules restored to a healthy state.

Be reserved, even in respect of children, in stating your diagnosis, when the patient has been suffering for a long time from severe catarrh accompanied by fever, if you have ascertained that bronchial blowing has been obstinately persistent at the same point for more than a month, and is accompanied by subcrepitant mucous râles, and does not depend on pleuritic effusion: be reserved in your diagnosis, for there is reason to fear that the child is a tuberculous subject.

Tubercular disease is more common during infancy and early childhood, than at any other period of life. Physicians who have had charge for a long period of institutions for infants at the breast know that most of their little patients die of tubercular disease of the chest. Unfortunately, the diagnosis of pulmonary tuberculisation is much more difficult in very young subjects than in others. Many of the elements which auscultation can alone furnish to enable us to form an exact opinion as to the existence of the characteristic lesion are absolutely wanting. The vesicular murmur, the anomalous sounds by which it is replaced, or accompanied, are heard with difficulty, as children often breathe badly, and never breathe (as adults do) in accordance with your directions. The same remark applies to vocal resonance; for, as I have already remarked, the resonance of the voice is in children replaced by the resonance of the cry. The same remark is also applicable to auscultation of the cough, which is so often an assistance in the stethoscopic examination of the chest. We cannot count upon the appearance of the sputa throwing any light upon the character of the disease, because, as a general rule, children do not expectorate.

If pulmonary tuberculisation be so difficult of diagnosis in the child, how much more difficult will it be in the child to establish

the differential diagnosis between tuberculisation and chronic pneumonia, inasmuch as it is in many cases almost impossible even in the adult to distinguish between the two.

I am aware that an attempt has been made to lay down characteristic signs, with a view to reach a solution of the difficulty. It is said that in the adult, the progress of the two diseases is different, and that purely inflammatory induration of the lung is generally the result of acute pneumonia, and that tubercular induration arises slowly, and seldom follows pure inflammation. The value of the first test is obviously open to dispute, because it is by no means unusual for an attack of pneumonia to determine the manifestation of the tubercular diathesis in the lungs, and leave behind it induration of specific character.

If the summit of one of the lungs is the chosen seat of tubercular induration, we can generally by attentive examination detect something on the opposite side. In chronic pneumonia, the lesion is only on one side, and is generally at the base or middle of the lung. It was otherwise, however, in the patient in St. Agnes's ward to whom I referred. In him, the lesion occupied that part of the lung, on the right side, corresponding to the infra-spinous fossa of the scapula, that situation in which it is so common to meet with tubercular engorgement.

The absence of hemoptysis in cases of chronic pneumonia, and their frequency in phthisis might furnish characteristic phenomena; but we know how often we discover tubercular induration in persons who have never spit blood.

General symptoms, such as rapid emaciation and night sweats, which supervene in tuberculisation and are absent in chronic pneumonia, are by no means unexceptionable differential signs, for it is not unusual to detect tubercles in their first stage in persons of apparently excellent health, and who only complain of a slight catarrhal affection: and we sometimes meet with others in whom there was nothing to arouse attention, but in whom, on careful examination, the presence of the serious and unsuspected disease was found. The resistance offered to the finger used in percussion, observed in chronic pneumonia as contrasted with the less complete dulness met with in tubercular induration, is another differential sign which has been mentioned; but it is one of so much delicacy, that I think it would be exceedingly difficult to prove its clinical value.

To sum up :—It is rather by induction, by an appreciation of the general character of the symptoms, by careful examination of the patient, by repeating the examination several times, and by watching the patient, that we can recognise the nature of his disease. We often learn more from the sequel of the case, and from supervening modifications in the phenomena recognised by auscultation and percussion, than from the previous history, or the facts we ascertained at the outset.

In the child, the differential diagnosis is still more difficult than in the adult. In the adult, coarse mucous râles, gurgling, cavernous blowing—the signs of the formation of a pulmonary cavity, by the softening of tubercular deposit—when they follow blowing sounds and subcrepitant râles, give ultimately almost complete evidence of the existence of the tubercular affection; but in the child, these signs will not afford you any absolute certainty, because the coarse râles, gurgling, and cavernous blowing may be signs of the small pulmonary abscesses which are common in the pneumonia of childhood, as well as of tubercular cavities. I repeat, however, what I have already said, that when you are consulted about a child who has been suffering for a long time from severe catarrh accompanied by fever, in whom you hear bronchial blowing which has been obstinately persistent for a month in the same situation, when the blowing is accompanied by subcrepitant mucous râles, and when you are sure that it does not depend on pleuritic effusion, do not pronounce an unreserved diagnosis; for there is reason to fear that the child is a tubercular subject. That is precisely the case of our little patient in the nursery ward. The duration of the symptoms for three months, and the persistence of the blowing heard upon his admission to the hospital, by making me at once reject the idea of acute pneumonia, and at the same time demonstrating the existence of chronic pulmonary induration, led me to the conclusion that there was tubercular deposit.

LECTURE XXXI.

GANGRENE OF THE LUNG.

Difficulties of Diagnosis.—Several Species of Gangrene of the Lung: One of them, the Species here more particularly considered, is Curable.

Gentlemen:—I have to speak to you to-day of a patient lying in bed 1, St. Agnes's ward. The pulmonary affection under which he is suffering has certain peculiarities which demand your earnest attention.

This man, about fifty years of age, has long been subject to attacks of catarrh, which have often proved violent and obstinate. He states that upon one occasion, some years ago, the attack was complicated with symptoms similar to the present. At the time he entered the hospital, some months ago, he was tormented by a frequent cough accompanied by catarrhal expectoration, which at first had nothing remarkable in its character in respect of the quantity and physical characters of the sputa. He was in a decidedly febrile state. In other respects, things went on with so much regularity as to give us no anxiety, when, quite suddenly, a few days after his arrival in our wards, he expectorated matter of so exceedingly penetrating a fœtor, that the nursing sister was obliged to keep the windows near his bed always open. All the patients in an adjoining ward, as well as in his own ward, complained of being poisoned by the horrible smell: and on one occasion, at the visit, I felt myself very much inconvenienced by his coughing. His breath and sputa diffused an insupportable gangrenous odour. After twelve, twenty-four, thirty-six, or forty-eight hours, the gangrenous odour was replaced by a sickly smell of honey, very disagreeable, and perhaps constituting a specific character of the disease.

These occurrences took place at intervals of a fortnight, eight days,

or even of only four days: they were sometimes accompanied by fever of greater or less severity, or perhaps there was no fever at all.

At each visit, I auscultated the chest with the greatest care, but I never heard gurgling, blowing, nor any sign of cavities in the lungs: I only heard sonorous rhonchus at the angle of the right scapula, and occasionally coarse mucous râles, which after being scarcely audible for twenty-four or forty-eight hours, all at once ceased. Percussion, however, elicited a very decided dull sound at the summit of the right lung, particularly behind.

In the absence of stethoscopic signs of softening of the pulmonary tissue, and of a cavity communicating with the bronchial tubes, I was naturally led by the characteristic odour of the sputa and breath, to think of gangrene of the lung: but the progress of the symptoms, their intermittence, and the predominance of the catarrhal element, also told me that I had to do with one of those special forms of gangrene to which Dr. Briquet first called the attention of practitioners, and regarding which I shall immediately speak.[1]

Gangrene of the lung has been rarely observed to follow pure pneumonia; and I have never seen a single case in which this has occurred. It was the opinion of Laennec that gangrene of the lung can hardly be placed among the natural terminations of pneumonia. But it may occur, when the pneumonia is of a septic nature. By a curious chance the only two cases of gangrene of the lung which I have seen, presented themselves to me in my wards at the Hôtel-Dieu within a fortnight of one another: the first occurred in a patient with malignant small-pox: the second, in a man with severe dothinenteria. I am not at present speaking of traumatic gangrene, to which attention has been directed, and of which you saw a case in bed 1, St. Agnes's ward. This man was operated upon for empyema, and recovered: I shall return to his case on a future occasion.

Laennec believed that gangrene of the lung is generally allied in its nature to affections which are essentially gangrenous, such as anthrax, malignant pustule; and, as in these affections, the inflammation developed round the gangrenous part seems to be the effect rather than the cause of the mortification.

Gangrene of the lung has been often observed in diabetic subjects:

[1] Briquet:—Archives Gén. de Médecine: 3me série, T. xi.

as you will see by the cases related by Griesinger, Monneret, Charcot, Marchal, and Fritz. There is here a sphacelas of the lung similar to that observed in the cases upon which Marchal (de Calvi) has justly laid so much stress.[1] The bad general condition of the system induced by diabetes, produces necrosis in the pulmonary passages, just as it produces the same effect in the limbs, and in the crystalline lens in cases of diabetic cataract.

Finally, I am inclined to think that pulmonary embolism may be the cause of more or less extensive gangrene of a portion of the lung, gangrene limited to the tissue in which ramify the branches and small ramifications of the obliterated vessel.

This was evidently the case in a young woman, whom some of you may remember, who, in October 1858 occupied bed 2, St. Bernard's ward. She had been recently confined, and after delivery had had *phlegmasia alba dolens*. One day, she suddenly complained of dyspnœa, and pain in the right side of the chest: the expectoration was very soon afterwards characteristic of the sputa in pulmonary apoplexy: and I entertained no doubt, that the pain in the side, the dyspnœa, and the apoplexy of the lung were the consequences of an embolus. Some days later, the sputa were characteristic of gangrene of the lungs. The patient sunk rapidly. At the autopsy, I found sphacelas of that part of the lung supplied by the vessel in which the embolus was situated. When an opportunity occurs of returning to the subject of embolism, I shall give you, *in extenso*, the details of this case. For the present, I have said enough to convince you that gangrene of the lung may be the result of an embolus in the pulmonary artery, although that vessel is not concerned in the nutrition of the organ. Afterwards, should an opportunity arise, I shall discuss this question in all its bearings; but at present, in support of the clinical fact, and to give it a more authoritative sanction, let me remind you, that Virchow, in his experimental researches, has fully recognised this cause of gangrene of the lung. He says:—"When the alterations produced by the embolus extend to the periphery of the lung, when the organ becomes gangrenous throughout a certain extent, the pleura itself sphacelates in the part corresponding therewith, and then ruptures, giving rise to pneumothorax." This is what took place in the young woman, our patient;

[1] Marchal (de Calvi):—Recherches sur les Accidents Diabétiques. Paris, 1864.

for besides gangrene of the lung, she had pneumothorax and gangrene of the pleura.

I shall not dwell on this kind of parenchymatous gangrene, the history of which was originally written in a complete manner by the author of the "*Traité de l'Auscultation Médiate :*" I shall only add, that among the causes predisposing to this affection have been mentioned excess in alcoholic stimulants, and inanition, the influence of the latter being very great. Indeed, gangrene of the lung is a pretty frequent cause of death in insane persons who have long refused to take food. Finally, let me remind you, that hæmorrhagic nuclei are frequently the starting points of this kind of gangrene; as is shown by the cases published by Dr. Genest,[1] and by a case, still more characteristic, communicated to the Anatomical Society of Paris by Dr. Firmin.

When I review the recollections of my personal experience, when I consult what has been written on this disease, I am struck with the inadequacy of the signs by which to determine the existence of gangrene of the lung.

The stethoscopic signs are at first nearly the same as those which we find in cases of pulmonary abscess: at a later stage, when the portions of sphacelated parenchyma have been eliminated, the physical signs are exactly the same as those which reveal the existence of a cavity in the substance of the lung, whatever cause may have produced the cavity.

The expectoration, though presenting something more characteristic, does not always furnish pathognomonic indications: it is only the odour which has a decisive import, for the aspect and colour of the expectoration is exceedingly variable, and often differs in no respect from the muco-purulent sputa of catarrh. The odour is sometimes absent at the beginning of the disease, and also at the end when there is a tendency to recovery. The peculiar gangrenous fœtor of the breath is the only pathognomonic sign of gangrene of the lung.

But even to this sign, we must not attach too much importance, as it may signally deceive. I have several times seen circumscribed pleurisy, and particularly interlobular pleurisy, give rise to symptoms simulating gangrene of the parenchyma. This occurs when perforation of the lung takes place. In a case of that kind, the pus

[1] Genest:—*Gazette Médicale de Paris.*

expectorated is small in quantity, and has at times a horrible fœtor, auscultation furnishing the signs of a limited cavity.

When speaking of dilatation of the bronchial tubes, I sufficiently insisted on the fact, observed by Laennec, that the pulmonary catarrhal affection sometimes assumes a strange fœtor, well fitted to lead to the belief that it is gangrenous fœtor. In certain persons, under the influence of violent inflammation, the secretion of the bronchial mucous membrane, like that from the nose, urethra, and vagina, has a disgusting smell, exactly like the gangrenous odour; but as I pointed out to you in our patient, the fœtor of the sputa, even when the gangrene is evident, notably differs from that which is observed in ordinary parenchymatous gangrene.

It is principally in the peculiar species of gangrene of the lung of which the patient, the subject of the present lecture, offers an example, that the diagnostic difficulties are greatest. Here, in fact, the signs furnished by auscultation and percussion differ in no respect from those which characterise catarrhal affections of the lungs, viz. mucous râles, bronchial blowing (sometimes amphoric), bronchophony, all the phenomena dependent on pulmonary catarrh, on dilatation of the bronchial tubes, or on small cavities. This arises from the fact, that in this particular species of gangrene of the lung, the affection does not involve the pulmonary parenchyma, but the extremities of the minute bronchial ramifications.

Here, in fact, are the anatomical lesions mentioned by Dr. Briquet in the two cases constituting the basis of his memoir in the *Archives Générales de Médecine* for 1841. The extremities of the bronchial tubes, dilated into pouches, form cavities on the surface of the lung, containing a viscid, greyish, very fœtid liquid: the pouches are lined internally by a very soft, flaccid, whitish membrane, which can be removed by scratching, and which exhales a strong odour of gangrene.

I was led to conclude that our patient had this particular form of gangrene from the great similarity which his symptoms presented to those of which my friend Dr. Lasègue has drawn the picture.[1]

A person of no particular age, of constitution more or less robust, a person generally speaking tried by previous hard work or much bad health, is seized with bronchitis, which at first has no special characters: the dyspnœa is not great, nor is the cough

[1] Lasègue:—Gangrènes Curables du Poumon. [*Archives Générales de Médecine*, 1857. T. ii.

severe: the expectoration is pretty abundant, such as it is in the advanced stage of bronchial catarrh. The general health, however, shows a change for the worse: the sputa become more profuse and more purulent, and sometimes their fœtor is such as to attract the attention of the patient and of those who are with him. This first critical period passes wholly, or to a great extent, without being perceived: the expectoration and the fœtor diminish or disappear, the bronchitis however, remaining: there is little or no fever.

After a period, varying in duration, the bronchitis seems to revive to a certain extent. The expectoration becomes greenish yellow, sometimes brown, or at other times grey; it again acquires a fœtor which is peculiar and gangrenous: it increases in quantity, and may become exceedingly profuse. Usually, it occurs in fits during the day-time, in the morning, in the evening, or during the night, leaving intervals of rest to the patient, during which the breath retains more or less of its disagreeable smell: strength diminishes, and there is less appetite: the digestive functions are not much disturbed, and there is little or no fever. On auscultation, we hear moist râles, coarse or subcrepitant, occupying a greater or less extent, persistent in some places, disseminated, mobile, accompanied or not accompanied by bronchial resonance of the voice, and without decided dulness; at times, there supervene rigors of short duration, followed by profuse spitting: the cough has no specific character. This state of matters may go on from weeks to months, and from months to years, to the great detriment of the general health, which, however, while it becomes feebler, does not reach that state of hectic debility characteristic of advanced tubercular disease: there is little or no hemoptysis. Notwithstanding the continuity of the malady, its activity is suspended from time to time, the expectoration diminishing, for whether the amendment is persistent or temporary, it always begins by a diminution in the expectoration: the fœtor gradually ceases, or suddenly disappears. During the intermissions, the stethoscopic signs become more faint, or are not at all changed.

If the patient get a long period of repose, he seems to become quite restored: but if his rest be short, the economy hardly derives any benefit. Whatever may be the course followed by the disease, from this point of view, the bronchorrhœa is always an essential fact. It is excess in quantity, rather than the nature of the expectoration, which seems to exert a prejudicial influence.

In the exposition now made of the phenomena which characterise this special form of gangrene of the lung, do you not find, Gentlemen, most of the symptoms complained of by our patient and observed in him by us?

Although presenting more than one point of resemblance to that form of gangrene of the lung which may be called the classical form, it essentially differs from the classical in being chronic in its progress, the other generally progressing in a more acute manner. It differs, too, in the predominance of the catarrhal element, in the expectoration being always very abundant, and consisting almost exclusively of mucus of a fœtid gangrenous odour; while in gangrene of the parenchyma, the sputa usually assume an altogether special appearance of animal detritus.

This form of gangrene differs from the other most of all in its being relatively a milder affection; for although parenchymatous gangrene has sometimes a propitious termination, it is evidently cases of the kind we have now been observing together which have furnished most of the examples of recovery.

The cures have generally been obtained by pulmonary atmidiatria. As you are aware, atmidiatria is a method of treatment which consists in administering medicines by the respiratory passages: it is sometimes practised with a view to obtain a general action on the economy, as when chloroform is inhaled to induce anæsthesia; or at other times, it is employed to modify an inflammatory state of the pulmonary apparatus.

Inhalations of the vapour of turpentine water, have been found of real service in cases of gangrene of the lung, by Professor Skoda of Vienna, who was the first to praise them. I used them in the case of our patient, employing Richard's fumigatory apparatus. This instrument consists of a tin vessel, into which water is put, and then heated by means of a spirit-lamp placed below it. Within this tin vase is a large glass flask, to which two tubes are attached, and which is filled with tepid water, kept, by means of this water-bath, at a temperature between 45° and 50° C. The temperature is regulated by a thermometer placed in one of the tubes: to the other tube is adapted a bent tube terminating in the form of the beak of a clarionet. The patient puts this beak into his mouth, and through it inspires air impregnated with the vapour of the water contained in the vessel, and charged with the medicinal substance. This instrument may now be replaced by the spray-apparatus of

Sales-Girons, of which I have spoken to you on more than one occasion.

The spray-apparatus allows, as you are aware, the vapour of the medicinal substances used to enter the deep recesses of the respiratory passages—not only the vapour of volatile substances, such as the essence of turpentine, the essential oils of cubebs and copaiba, which may also be administered by Richard's instrument, but it enables us to introduce into the lungs non-volatile therapeutic agents, provided they are soluble in water. In the form of gangrene of which I have now been speaking, I have also made use of preparations of tannin, of solutions of the extract of rhatany, of sulphate of copper, of corrosive sublimate, of arseniate of soda—powerful modifiers, which when introduced into the bronchial tubes act upon the diseased surfaces in a manner wonderfully conducive to recovery. I need not say that at first the solutions used must be exceedingly weak, and that their strength requires to be slowly increased in proportion to the increased tolerance of the economy.

LECTURE XXXII.

PLEURISY:—PARACENTESIS OF THE CHEST.

PLEURISY.—*Ordinary Signs.—Skoda's Bruit.—Interpretation of the Rubbing Sound.—Crepitant Râles of Pleurisy. Persistence of Blowing Sound in Cases of Excessive Effusion.—Blowing Sound, and Amphoric Voice, are Signs of Pleurisy.—Mistakes in Diagnosis may sometimes occur.—Intercostal Fluctuation.*

GENTLEMEN:—I readily admit that in the immense majority of cases pleurisy is an easily recognised disease. In proof of the correctness of that statement, I only require to remind you of the signs of the disease given by all your classical authors, and to which I never cease to direct your attention at the bedside of the patient. The stitch in the side, the cough, the absence of expectoration, the obscurity and then the dulness of sound in the parts most dependent, the increased volume of the chest on the affected side, the absence of thoracic vibration and respiratory murmur, the blowing sound, the egophony, the bronchophony, and other signs are familiar to you.

Nevertheless, in some cases, fortunately very exceptional cases, all the signs of pleurisy exist, and yet the autopsy reveals a different lesion. Quite recently, my colleague in the service of the hospitals, Dr. Empis, found all the signs of pleuritic effusion in a young woman, received into his wards at La Pitié, with pain in the right side, dyspnœa, and fever. Percussion elicited absolute dulness in the two inferior thirds of the right side of the chest: by auscultation, it was found that in the lowest part of the right lung there was an almost total absence of respiratory murmur, while in the two middle thirds, both before and behind, there was very loud bronchial respiration, accompanied by a considerable amount of egophony. The patient died; when it was discovered that the case was one of encephaloid tumour, and that there did not exist the least effusion of fluid. Two years ago, the same physician communicated to the

Medical Society of the Hospitals a curious example of hydatid cyst of the liver, which had pushed up the diaphragm and the lung in such a manner as to occupy the two inferior thirds of the right side of the chest, and so given rise to the signs of pleuritic effusion, though there was no effusion. I shall now quote the account of this case, which has been published by Dr. Empis.

"When Dr. Monneret entrusted me with his wards, on leaving town for the holidays, he told me that the patient whose case is the subject of the following history was suffering from profuse pleuritic effusion, to effect the removal of which he had in vain exhausted the resources of medicine, and for which he thought paracentesis of the chest was indicated. He added, that he had attempted the operation some days previously, but having, as he had thought, made the puncture too low down, he had not given exit to any fluid, and had, he believed, penetrated the liver. He requested me to repeat the operation, and to make the puncture a little higher up. The existence of a pleuritic effusion did not seem to be a matter of doubt. There was bronchial blowing, and egophony, than which nothing could be more characteristic, at the junction of the superior third with the lower part of the chest. The patient was in a cachectic state, and was sinking day by day. I concurred in Dr. Monneret's opinion that thoracocentesis was indicated. M. Regnault, the *interne*, performed the operation in my presence, introducing the trocar between the fourth and fifth ribs: greenish pus immediately issued from the canula in such quantity as to fill the basin: then, almost of a sudden, this flow of pus ceased, and could not be re-established. The stethoscopic signs were little changed: bronchial blowing and egophony could still be heard, and the dulness had not diminished in proportion to the quantity of fluid which had been drawn off. We left the patient quiet for two days and then gave him an emetic: he brought up a great quantity of pus. The pus had evidently made a way for itself through the lung. Soon afterwards, the patient died. At the autopsy, we found that there was no trace of effusion into the pleura; and that the disease was a large hydatid cyst of the liver which had suppurated, and which, from the enormous size it had acquired, had crushed up the diaphragm and the lung into the upper third of the chest, and so occasioned the dulness and the signs of pleuritic effusion which have been described. This case proves that bronchial blowing, egophony, and dulness are not always sufficient signs of effusion

into the pleura, seeing that they may be produced by fluid encysted below the diaphragm pushing up the lung, and remaining in contact with it."[1]

I have spoken to you a hundred times of the modifications which the signs of pleurisy may undergo in various patients compared with others; and in the same patient, according to the stages of his disease, as well as according to the quantity and nature of the effusion. I do not wish to go back upon these points to-day, and shall limit my remarks to some new signs, certain of which are universally accepted, while the value of others is still under discussion.

Some years ago, Gentlemen, the peculiar thoracic resonance described by Skoda [*retentissement skodique*] was recognised by very few physicians: at the present day, it is generally admitted, that in pleurisy, on percussing below the clavicle and in the region nearest to the sternum, there is heard a peculiar semi-tympanitic sound, to which my illustrious colleague of the Vienna School was the first to draw the attention of observers.

It is quite true that in some exceptional cases, where it is evident that there is only pneumonia, Skoda's resonance may be produced, as I have repeatedly pointed out to you at the bedside. Other physicians, among whom is Dr. Woillez, have arrived at the same conclusion with me on this point; but this sign is almost never absent in pleurisy, when the effusion does not come up above the fourth rib, and it is only met with exceptionally when pneumonia exists alone, uncomplicated with pleurisy.

I must, however, Gentlemen, somewhat limit the statement I have now made. I have told you that a patient may have pleuritic effusion, although by the most attentive examination we cannot hear blowing, egophony, or bronchophony; and when no other signs can be discovered, except dulness and absence of the respiratory murmur. I lately received in bed 6 St. Bernard's ward a woman, who, along with serious lesions of the heart, had pleuritic effusion on the right side. Not finding the usual signs of the disease, I carefully practised auscultation every day, but I never once heard blowing, egophony, or bronchophony. Although there were no other physical signs than dulness, with absence of vibration and respiratory murmur, I had no hesitation in affirming that there was effusion.

[1] Empis: — Bulletin de la Société Médicale des Hôpitaux (Séance du 9 Octobre, 1861).

At the autopsy, I found so great an amount of serous fluid in the pleura, that I exceedingly regretted not having performed paracentesis of the chest.

You are aware that the friction sound has been considered as a precious diagnostic sign of pleurisy. At the beginning of an attack, before any effusion has taken place, or while the quantity of fluid is as yet very small, the sound is supposed to be produced by the respiratory movements causing a rubbing upon one another of the two folds of pleura, the surfaces of which are covered with a thin layer of fibrinous exudation. Towards the close of the pleurisy, when the diminished quantity or complete absorption of the effused fluid permits the two pleural surfaces to come into contact, the friction sound is attributed to their being coated with false membrane, more or less thick and resisting.

Gentlemen, the real friction sound of pleurisy is much more rare than is generally said and believed.

I have seldom had an opportunity of hearing it at the beginning of a pleurisy, a circumstance sufficiently explained by the fact, that I am seldom called in at that early stage of the disease; and that a few hours are sufficient to allow a more or less considerable effusion to take place. It is generally towards the end of the attack that we have the best opportunity of recognising the sound produced by the rubbing of the pleural surfaces. I again repeat that this friction sound is much less common than has been alleged.

I wish to put an end to any misunderstanding with the physicians who do not concur in that opinion.

In the first place, the kind of sound which is heard at the beginning of an attack of pleurisy, resembling the rustling produced by the friction of two sheets of very fine crisp paper, and to which the name of friction sound [*bruit de frottement*] has been given, is in my opinion a blowing sound. I base this opinion on the following considerations. If you auscult your patient twice or thrice a day you will find that this alleged friction sound, becomes more and more harsh, till at the end of twenty-four or forty-eight hours, it has become a true blowing sound [*véritable bruit de souffle*] such as may be heard in pneumonia. The voice at the same time has a distinct egophonic resonance, and, in proportion to the degree in which the friction becomes decided, the voice passes from a bleating bronchial resonance to a pure bronchial resonance [*à l'égobronchophonie, enfin à la bronchophonie la plus nette*]. I am consequently

justified in declining to call the sound in question a friction sound, and to regard it, with many other clinical observers, as a modification of bronchial blowing.

The friction sound heard in the decline of a pleurisy also demands a few explanatory words. Quite at first, when coexistent with the pleurisy, there is pulmonary emphysema or chronic bronchitis, we sometimes hear vibrating râles, which continue audible for a long time in the same part of the lungs, and which resemble so much, as to be liable to be mistaken for, the peculiar sound produced by rubbing the point of the finger on the hand when the skin is dry, or by pressing a bit of snow between the fingers; but if this sound continue at a determinate part of the lung, particularly in the anterior, middle, and lateral regions, if it continue to be found after we have made the patient cough and expectorate, it is hardly possible to confound it with a sonorous râle—it is then the friction sound, the existence of which I never had the least intention to deny.

Again, there is a sound of another kind which is heard at the end of a pleuritic attack, which has also been regarded as a friction sound: it resembles fine crepitation, and is a very different sound from that about which I have been speaking. This sound, which is met with in the great majority of cases of pleurisy is, in fact, a crepitant râle; and I have called it *the crepitant râle of pleurisy*. My interpretation of it is very simple. Just as we never have erysipelas without engorgement of the cellular tissue, there cannot be erysipelas of the pleura, or pleurisy, without an irritative engorgement of the subpleural cellular tissue or of the peripheric pulmonary parenchyma. This fluxion naturally carries with it into the pulmonary vesicles a serous exudation analogous to that of pulmonary œdema. We also meet with a fine subcrepitant râle, which is very often heard quite at the beginning of the pleurisy, and which likewise nearly always continues for some weeks, when, the fluid being absorbed, there only remains sub-inflammatory œdema of the more superficial parts of the lung.

I must not forget to mention a sign to which I have already often called your attention:—I refer to the persistence of bronchial blowing and bronchophony in cases of excessive effusion. I had long believed, on the statement of my teachers, and of the best authors on the subject, that the blowing disappeared when the effusion became considerable; but after I had many times performed the operation of paracentesis of the chest, I became convinced that not

unfrequently, in cases in which the effusion amounts to several litres, and when the dulness extends up to the clavicle, when the diaphragm is pushed out of place, and the intercostal spaces dilated, bronchophony and the blowing sound continue up to the very moment at which the trocar affords an exit to the fluid. You recollect that I have often invited you to ascertain for yourselves the presence of this sign; and you also had an opportunity of observing, in the same cases, that when the puncture was made, there was a large quantity of fluid evacuated.

Gentlemen, in cases of pleurisy, we often meet with all the stethoscopic signs which belong to the third stage of tubercular phthisis. The attention of practitioners has been particularly called to this important point in diagnosis by Drs. Rilliet and Barthez,[1] Dr. Béhier,[2] and (more recently) Dr. Landouzy.[3] It is now a recognised fact in medical science; and if—as I am about to tell you—there is still a risk of committing great mistakes in diagnosis, it is not the less incumbent on us to bear in mind that till the publication of the researches of the physicians whom I have just named, this curious point in the history of pleurisy had not been well studied.

Amphoric respiration, gurgling, and cavernous voice are sometimes so well marked, that it is impossible to avoid attributing them to the existence of cavities in the lungs, particularly when the sounds emanate from the summit of the lung; and even when they are heard towards the inferior angle of the scapula, the same idea presses itself upon us, so identical seems the gurgling and blowing with similar sounds proceeding from large excavations in the centre of the pulmonary parenchyma.

But nevertheless, the mode in which the disease commences and progresses, the dulness of the dependent parts, the displacement of neighbouring organs, the volume of the chest, the absence of lesions

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[1] Barthez et Rilliet:—Sur quelques Phénomènes Stéthoscopiques rarement observés dans la Pleurisie Chronique.' [*Archives Gén. de Médecine;* March, 1853.]

[2] Beheir:—Note sur un Souffle Amphorique observé dans deux cas de Pleurisie Purulente Simple du coté droit. [*Archives Gén. de Médecine;* August, 1854.]

[3] Landouzy:—Nouvelles Données sur le Diagnostic de la Pleurisie et les indications de la Thoracocentèse. [*Archives Gén. de Médecine;* November and December, 1856.]

at the summit of the lungs, in a word the general condition of the individual, usually enable us to form a diagnosis. However, it is sometimes difficult to avoid error. In the memoir of Dr. Barthez and Rilliet, you will find a very interesting case in which a mistaken diagnosis was formed by a very experienced physician.

In acute or chronic pleurisy, what are the conditions which give rise to gurgling, and to amphoric breathing, voice, and cough? Drs. Barthez and Rilliet, recollecting that in a case of pleurisy complicated with pneumonia they had observed increase of the bronchial blowing, were led to think that in chronic pleurisy with bronchial respiration of cavernous tone, there existed, along with the effusion, more or less induration of the pulmonary parenchyma. Dr. Béhier says that the amphoric sound is not heard in cases of effusion into the pleura, unless the lung, compressed and indurated, is in contact with the trachea or one of the large bronchial tubes. We can understand that the laryngeal sounds may assume an amphoric tone by transmission through indurated lung and through effusion compressing tubes of large calibre, such as one of the chief divisions of the trachea: then again, if the trachea or bronchial tubes contain a certain quantity of mucous secretion, the beating about of that fluid by the air will produce gurgling. This explanation given by Dr. Béhier, and not very different from that formerly furnished by Drs. Barthez and Rilliet, is applicable to some cases, but not to all.

It appears that when extensive effusion takes place into the cavity of the pleura, the lung, pushed up to the top of the chest and into the hollow of the vertebral column towards the root of the bronchi, is in a condition favorable to the production of amphoric sounds; but that nevertheless they are not always produced. It also appears, however, that amphoric sounds may be produced when the effusion is so small in quantity as to allow the lung to retain very nearly its normal relations. The case which I am now going to describe afforded you an opportunity of verifying this statement for yourselves.

On the 14th April, 1862, a woman aged twenty-one, became the occupant of bed 30, St. Bernard's ward. She had been confined at the hospital of Lariboisière on an early day in November 1861; and a few days after delivery, she had had some affection of the right side of the pelvis. This affection could not have been serious, for she was able to leave the hospital fifteen days after the birth of the child.

From that time, she had fever and vomiting at each menstrual period. On the first occasion of my examining her, I discovered a large tumour in the right iliac fossa: it reached up, on both sides, to the crest of the ilium, and descended to the lateral parts of the pelvis, posteriorly enveloping the uterus, which was enclosed by it. The tumour, which seemed to me to be a pelvic abscess, very slowly diminished in size, and in a month had nearly disappeared. As it did not occasion any unfavorable symptoms, it was let alone, so that I might attend to the patient's other, and much more important, pathological conditions—conditions upon which I now wish to fix your special attention.

On the 18th April, that is to say four days after admission to the hospital, our young patient complained of pain at a particular spot of the left side. Auscultation did not disclose to us any signs other than those of acute general catarrh. The chest symptoms seemed to be improving, when on the 29th April, that is to say, nine days from the beginning of the bronchitis and the stitch in the side, she showed evident signs of pleurisy on the right side; and at the same time we detected an obscure blowing sound and subcrepitant râles over the angle of the left scapula. We had, therefore, a case of double pleurisy, complicated with slight bronchopneumonia. During the following days, the signs on the right side, which were those of pleurisy, viz. absolute dulness, bronchial blowing at the base, egobronchophony within the limits of the situation occupied by the effusion, became more and more decided: on the left side, I only found egophony along the vertebral column; but at the same time, I heard gurgling, and moist crackling such as is observed at the summit of the lungs when full of softening tubercular masses.

While the stethoscopic signs on the left side remained without any sensible change, those which characterised the effusion on the right side became more and more decided. Paracentesis was resolved upon. The operation was performed by Dr. Dumonpallier, who followed the rules and observed the precautions which I have long ago laid down. Nine hundred grammes [about a quart] of a purely limpid serosity were withdrawn. The operation was followed by a great amelioration in the state of the patient; but the serous effusion rapidly reaccumulated, and in four days it was necessary to tap a second time. It was the last time; for there was no recurrence of the effusion: the dulness, however, continued up to the

end, and eight days before death, there was heard slight egophony near the angle of the right scapula. A few days after the operation, a new morbid cardiac sound was heard.

On the 4th May, I began to hear distinctly on the left side, principally near the angle of the scapula, a blowing sound, amphoric respiration, and amphoric gurgling. From day to day, the amphoric, cavernous gurgling and respiration were objects of attention: they continued to be heard up to the death of the patient. Each day, five or six persons verified the stethoscopic signs. Dr. Landouzy of Rheims, who was at the time on a few days' visit to Paris, honoured me with his presence at the visit; and after examining the patient, had no hesitation in concurring with my opinion, to the effect that bronchitis and pleurisy existed on the left side. Pay particular attention to the fact, that while we heard mucous and subcrepitant râles in the middle and lower parts of both lungs, nothing of the kind could be observed in their summits.

Upon comparing the signs furnished by auscultation and percussion in the case of this young woman, with the signs found in phthisical subjects, it will be seen, that there is no difference in the seat of the sounds. The expectoration was always that of bronchitis: there were no sputa coming from large cavities, nor was there any expectoration from pulmonary or pleural vomicæ. The effusion was apparently diminishing, the dulness was not complete, and was non-existent above the inferior angle of the scapula, but the amphoric blowing and the gurgling continued in the dependent part of the lung, and along the vertebral column. The patient very soon had œdema of the extremities and puffiness of the face; but the urine did not contain any albumen. She rapidly lost strength from intractable diarrhœa. The oppression in breathing became greater; and up to the evening before her death, the amphoric respiration and gurgling were heard in the situation already described. I shall at present only notice the anatomical lesions which were found in the lung. The right lung was, in its whole extent, adherent to the costal pleura: there was no trace of tubercle; and the bronchial tubes were filled with muco-purulent secretion. The left lung was elastic, free from all adhesions, devoid of tubercle and all other abnormal deposits. On making a section of the lung, muco-purulent matter flowed from the divided bronchial tubes. The sac of the pleura contained from 300 to 400 grammes [nearly a pint] of yellow serosity unmingled with fibrinous deposits: there was no false mem-

brane on the surface of the lung. The pleural effusion then was inconsiderable, and the lung was hardly at all compressed, although the amphoric blowing had been heard on the evening preceding death.

It must, therefore, Gentlemen, be accepted as an unquestionable fact, that amphoric blowing may exist without the lung being indurated or crushed up in a mass [*sans tassement*]; without there being any adhesions, or any pseudo-membranous deposits on the pleura; and finally, without there being any compression of the large bronchial trunks. The gurgling sound, the seat of which was in bronchial tubes filled with muco-purulent secretion, was transmitted to the ear by superficial compression of the lung in the situation of the pleural effusion. It appears then, that other conditions besides those mentioned by Drs. Barthez, Rilliet, and Béhier may give rise to amphoric sounds.

It is curious to observe what takes place during the flow of the serum from the puncture made by the trocar, as well as what occurs subsequent to that operation. Proportionately to the escape of the fluid, the displaced organs resume their normal position; the chest tends to return to its natural shape; and very soon the play of the ribs and diaphragm is again seen. Skoda's resonance ceases, and the lung expands: although dulness is persistent over the greater part of the chest, we can discover, on applying the ear, râles of variable volume, râles deep seated and distant; and frequently, it is not till after the lapse of some time that the blowing and egophony disappear. Let me now state what is generally observed in cases of acute effusion, when the puncture is made ere the lung has contracted adhesions, and before it has become encased in thick, resisting, undilatable false membrane. In effusion of old date, the lung is enveloped in thick false membrane, the thoracic walls are immovable, and it is only the displaced abdominal organs which regain their normal position consequent upon the draining off of the fluid: the lung, impermeable to air, remains fixed in the vertebral hollow: râles are no longer audible: if blowing and egophony existed before the puncture, they remain, and are sometimes louder than previously. The enunciation of these phenomena, which are described in a memoir by Dr. Landouzy, show, that effusion of fluid into the pleura has not in itself the power to produce blowing sounds and tremulous voice. I am very far, therefore, from professing Laennec's theory, a theory accepted by all, which attributes the metallic tinkle, Punch-and-

Judy tone, or goat-like sound of the voice, to the presence of more or less fluid in the pleura. But is it correct to conclude that the effusion has no part in producing bronchial blowing, amphoric blowing, and egophony? No: but the effusion operates in the production of these sounds only by compressing the lung, condensing it, so rendering it a better conductor of the sound produced in the bronchial tubes, or only transmitted by them and the trachea. The fluid, consequently, acts in the same way as false membrane, by tightly squeezing the lung, so that, in extreme cases of effusion, the blowing, as well as the egophony, are sometimes persistent. Let me remark, however, that bronchophony is more usually observed in these cases, and that it is the almost necessary companion of bronchial blowing. Egophony, with its different modalities, is, nevertheless, a very valuable diagnostic sign in cases of moderate pleuritic effusion.

Gentlemen, while in many cases of pleurisy, we find all the signs which belong to the third stage of tubercular phthisis, and while under such circumstances an error in diagnosis is excusable, it also sometimes happens that patients presenting all the signs of that form of pleurisy on which Drs. Rilliet, Barthez, Béhier, Landouzy, and I have so much insisted are really phthisical subjects, in whom the disease is localised in the middle and lower parts of the lung, and in whom there likewise exists chronic phlegmasia of the pleura, which is necessarily present when the tubercular lesion is in a very advanced stage.

You no doubt distinctly remember a young woman who lay in bed 28 of St. Bernard's ward, with whose case our attention was particularly occupied for a whole month. I shall now, in a summary manner, state the history of her case.

The patient, eighteen years of age, who had been subject to cough for two months, but had only been confined to bed for a fortnight, was admitted to St. Bernard's ward on the 23rd May, 1863. The disease began like bronchitis with severe catarrh. There had been no hemoptysis. The fever was slight. The expectoration was mucous and scanty. There was a little pain on the right side of the chest. Diarrhœa, accompanied by abdominal pain, had existed for a fortnight.

The resonance of the chest was normal on the left side, both at base and summit; but on the right side, there was complete dulness posteriorly of the two lower thirds, the resonance of the summit

being good. Auscultation of the left side only revealed vibrating or mucous râles disseminated in the middle and inferior but not in the upper part of the lung: on the right side, at the summit, there were no signs more marked than those found in the corresponding region on the left side; but in the situation where there was dulness on the left side, that is to say about the middle of the chest, there were coarse râles, blowing sounds, and amphoric voice.

The diagnosis was—general bronchitis, and pleurisy on the right side with amphoric sounds. This diagnosis was based on the progress of the disease, on the nature and small quantity of the expectoration, and on the seat of dulness and abnormal sounds.

For a fortnight, matters remained in this position; and at the end of that period, there was a marked increase of fever and diminution of strength. Mucous râles were heard at the summit of the left lung: but it was in the centre of the lung that the râles were most numerous and the bullæ least voluminous: towards the base, they were as stationary as in capillary bronchitis. On the right side, the respiration and the voice were amphoric.

From the twentieth day after her admission, she had a paroxysm of fever every evening: her general state became very bad: her aspect was that of an individual with typhoid fever: she had headache, noises in the ears, deafness, and giddiness when standing upright. Her tongue was dry, and she had urgent thirst. She had passed her motions involuntarily for two days.

On the 25th day, I heard on the left side, and for the first time, gurgling and amphoric blowing in the subspinous fossa of the scapula: some râles had an almost metallic resonance. The râles continued to predominate at the base, towards which situation they were more numerous, finer, and nearly crepitant. In the middle of the right side of the chest, towards the vertebral hollow, there were still heard the blowing sound, râles, and amphoric voice. The expectoration remained mucous and scanty: in the twenty-four hours, she only filled a fourth part of her spittoon.

Death, which had been foreseen, in consequence of the rapid wasting of the body and the intensity of the fever, occurred on the 22nd June, exactly a month from the date of the patient's admission to our wards.

At the autopsy, there was found general tubercular peritonitis, and ulcerations of the intestine, which explained the persistent

diarrhœa. During life, the existence of these lesions was not indicated by any pain in the abdomen.

There was very little fluid found in the right pleural sac, although the thoracic pleura was injected with blood and studded with crude tubercle. There were no pulmonary adhesions. The two inferior thirds of the right lung were transformed into an almost compact mass. In the vertebral hollow, the situation in which the amphoric sounds had been heard, there was a tubercular mass, the size of a small orange, presenting the aspect and consistence of mastic. In its most superficial part, there was hollowed out a cavern the size of a small filbert nut, separated from the surface of the lung by a partition which at most did not exceed two millimeters in thickness: in the neighbourhood of the cavity now described, there were five or six smaller ones in course of formation. A tolerably large cavity and three smaller ones were found at the base of the lung, which was bound to the diaphragm by close and almost cartilaginous adhesions. The summit of the lung was supple and crepitant. When cut into, the section showed tubercular granulations which at certain points were joined together in twoes or threes. Around the tubercular masses, the pulmonary tissue seemed healthy and perfectly permeable to air.

At the middle of the upper lobe of the left lung, there was found a cavity large enough to contain a hazel-nut: around this cavity were three much smaller cavities, which accounted for the cavernous râles with metallic resonance heard during the latter days of life. Throughout this lobe, numerous crude tubercles were disseminated. We found pulmonary obstruction, and concomitant bronchitis. The inferior lobe was everywhere pervaded by tubercles: in its superior part, there were crude tubercles, and at the base grey granulations.

The patient, though a woman in respect of development of organs, was a child when regarded from a pathological point of view. She had the tuberculisation of childhood—generally disseminated and not circumscribed tuberculisation—the acute and not the chronic form of the disease. Not only had she tubercles almost everywhere; but the pulmonary tuberculisation exhibited that irregularity in localisation which is peculiar to childhood, that is to say a development of the disease proceeding sometimes from the base to the summit, and not always from the summit to the base as in the adult. The result was an occurrence, quite exceptional in a woman, though perfectly usual

in a very young child—the formation of cavities at the base of the lungs before tubercles had appeared in their summits.

You now understand how, taking into account the rarity of cavities being produced at the base of the lungs in the adult, when none exist in their upper parts, and on the other hand, considering that pleurisy with signs of cavities, though in itself unusual, is more frequently met with than that rare form of tubercular disease, it was more rational to conclude that the young woman had pleurisy than tuberculisation.

Gentlemen, I have still a few words to say regarding *intercostal fluctuation*, a sign which appears to me to have a certain degree of importance, because, in cases of effusion into the pleura, it confirms the information furnished by thoracic dulness. Surgeons have pointed out that fluctuation in the intercostal spaces is met with in cases in which purulent effusion has formed an exit for itself through the thoracic walls; but I am not aware that intercostal fluctuation has been described as a sign of pleural effusion. Let me explain to you how I was led to suspect, investigate, detect, and finally to produce at pleasure this special fluctuation.

In practising percussion, I had for a long time been in the habit of employing a pleximeter and a hammer. When measuring, in my hospital patients, by striking on the pleximeter, the extent of the dulness discovered, the hypothenar region of my left hand resting on the wall of the chest, I felt that at each stroke of the hammer an impulse was conveyed to the pleximeter. With a view to ascertain whether or not the vibrations were imparted by the hammer to the chest, and transmitted by the ribs, I so placed my hand that its hypothenar region chiefly rested on an intercostal space; I thought I then felt fluctuation. Placing the palmar surface of my index finger upon an intercostal space (I percussed between different intercostal spaces) I distinctly felt fluctuation at each stroke: by making repeated experiments, I was easily able to determine the difference between the vibrations transmitted by the thoracic walls and by the fluctuation of the fluid. In thoracic vibration, there was felt under the hand the vibration of a mass, whereas on applying the palmar surface of the index finger to an intercostal space, the sensation was that of a fluctuating liquid. This fluctuation which many of you have been able to verify along with me is not at all a matter of doubt, when the observer proceeds in the manner I have now described. By a little practice one easily acquires the art of detect-

ing the fluctuation. I must add that it is not easily perceived, when there is a large amount of effusion. I do not wish to attach too much importance to this sign; but I think it is one which deserves to be mentioned.

Before speaking to you of paracentesis of the chest, an important subject which will occupy us during several meetings, I was anxious to discuss some of the new questions connected with the diagnosis of pleurisy. It will be easier for me to lay before you what I have to say upon puncturing the chest, now that I do not require to digress from my principal subject, to explain details regarding the diagnosis of effusion.

Paracentesis of the Chest.—Cases.—Historical Sketch of the Operation for Effusion into the Cavity of the Pleura.

In 1855, I performed paracentesis of the chest in a female patient of thirty years of age who had pleurisy with extensive effusion. The woman to whom I refer was the occupant of bed 12, St. Bernard's ward. She had always enjoyed good health. At least, she said, and repeated many times, that she had never had illness in any degree serious, till attacked by the malady on account of which she came to the hospital. The beginning of the malady she thus described.

About two months before her admission to the Hôtel-Dieu, without any preceding discomfort, without exposure to cold, without appreciable cause of any kind, she was suddenly seized during the night with exceedingly violent pain in the side. Next morning, however, she went to work as usual, although there was still some pain, which was increased by the smallest exertion. The breathing was oppressed, and much shorter than usual. For seven weeks, the only constitutional symptoms which showed themselves were slight general discomfort and loss of appetite; but eight days later, they had so greatly increased in severity, that she was compelled to relinquish her ordinary avocations, to keep her room, and indeed her bed, for the greater part of the day.

She had rigors on the 2nd May. On that day, her difficulty in breathing, till then not very great, became more urgent, and during the afternoon, she was admitted as a patient to the Hôtel-Dieu. During the evening, she was seen by Dr. Beylard, my *chef de clinique*, who found her in a feverish state. He noted the following particulars.

On uncovering the chest, he was at once struck with the conspicuous thoracic deformity. The left side was greatly arched; the left was more flattened than the right subclavicular region, and during great inspiratory movements, the left side did not appear to move. On percussion, absolute dulness was detected, extending from below upwards in front, till within four or five centimeters of the clavicle, and behind, to the crest of the scapula. Above the region of dulness, both in front and behind, there was resonance. On applying the ear to the chest in that situation, bronchial blowing and bronchophony were heard. The blowing extended to the top of the scapula; and in the infraspinous fossa well marked egophony was heard.

The patient had slight cough without expectoration. There was not much fever.

The diagnosis was easy. It was evidently a case of extensive pleuritic effusion, produced by one of those singular pleurisies which are accompanied by slight general symptoms, and yet lead to very profuse serous exudation.

This case, Gentlemen, merits all your attention, as a proof that there is a species of pleurisy, in which, if we are to form an opinion from the general symptoms, inflammation has little to do, and in which the functional disturbance dependent upon the lesion of the respiratory apparatus is so insignificant as to escape notice. In ordinary acute pleurisy, along with fever and other decided symptoms of constitutional disturbance, there is a violent stitch in the side and great dyspnœa: but in that particular form of pleurisy of which I am now speaking, there is hardly any fever, the stitch in the side is scarcely felt, and respiration seems to go on as usual. Well, then, Gentlemen, mark, that it is this species of *pleurisy*, to a certain extent *latent*, which gives rise to the most profuse effusion. The constitutional disturbance, I repeat, caused by the effusion is apparently so insignificant, that the individuals allow a very long time to elapse before they seek medical aid; and consequently, the physician has only the signs furnished by auscultation and percussion to guide him in his diagnosis.

Our patient consulted two physicians; and one of them, to whom she applied for advice for a uterine affection, did not even suspect that she had effusion into the pleura, because he saw that she had come to his house on foot, and did not make the slightest complaint of being winded from ascending his stair. Clearly understand, Gentlemen, that in mentioning this circumstance, I do not reproach

my colleague for having committed an error in diagnosis; for he certainly did not make an error: I only wish to show you, how easy it is to allow an affection to pass unperceived, when it does not declare itself, and when its physical signs have to be searched out.

I remember that in 1845, a nurse came on foot, carrying her infant, from the Pointe Saint-Eustache, where she lived, to the Necker Hospital, where I was then one of the physicians on duty. She had walked that distance, about four kilometers [2½ English miles] without being much tired. The effusion was nevertheless so considerable, that on the very day she came into our wards, I judged it indispensably necessary to perform paracentesis. I withdrew 2,500 grammes [two and a half litres] of fluid. This woman certainly seemed to be very little of an invalid: and so slightly did she feel out of sorts, that on the evening before the day on which she came into the hospital, she was working as usual.

The absence of oppression in breathing is a very important feature for consideration in these cases: I cannot too earnestly impress on your minds what I have now described, and what you yourselves have seen in our patient of St. Bernard's ward. Although her chest contained two litres and a half of serous effusion, her breathing seemed to be scarcely affected. Treasure this fact in your memories, for dyspnœa has been given, and indeed formerly used to be given by myself, as the chief indication of the necessity of paracentesis. I was singularly mistaken as to its value, as I shall have occasion to tell you in the course of these lectures. To wait for the dyspnœa, as has been recommended, and as was formerly laid down by me as the rule, is to run the risk of allowing the time for operating to pass, and of letting the patient die, as I have done. It is above all things important, to ascertain the extent of the effusion. Upon this point, auscultation and percussion furnish us with information in which we can place implicit reliance. The chest must be examined daily by these means; and when the progress of the hydrothorax is watched in this way, and is seen to be increasing very rapidly, the indication to operate is peremptory, whatever may be the degree in which dyspnœa exists—whether there be great difficulty in breathing, or whether there be no difficulty at all. It was this consideration which constrained me to operate at my first visit without any waiting, in the case of our patient in bed 12, St. Bernard's ward.

On the evening of that day, as I have already said, Dr. Beylard had still found resonance in the infra-spinous fossa, and in a space of from 4 to 5 centimeters under the clavicle. Next morning, the dulness was absolute everywhere. The effusion, therefore, had made great progress within 15 hours. It was estimated that half a litre of liquid was secreted by the pleura between morning and evening. Moreover, the displacement of organs testified to this increase.

The heart was not in its natural position; the apex was felt to beat under and near the right edge of the sternum, as was easily ascertained by the aid of the stethoscope and plexiметer. That I might eliminate every source of error, and not be influenced by the sense of sight, I percussed the patient with the eyes shut. Proceeding thus, I limited the dulness, beginning at the right, to three centimeters beyond the median line: the mediastinum and heart, therefore, were considerably displaced, and pushed to the right; on percussing from above downwards, I found dulness extending from the border of the false ribs, where I discovered that the spleen was out of its normal situation, showing that the diaphragm was squeezed up.

The great amount of effusion, and its rapid progress within a short space of time, convinced me that the operation was urgent, and could only be delayed at the risk of the patient dying before next day. I performed paracentesis of the chest according to the plan I have described to you, and drew off 2,000 grammes, (2 litres,) of perfectly clear yellowish serosity. As the fluid was being evacuated, the patient experienced a measure of comfort which was a great contrast to the feelings of distress of which she had previously been complaining. The vaulted form of the chest was gone; and with the aid of the plexiметer, I could follow the movements of the heart, and perceive that its apex was again in its proper situation near the left nipple. The spleen too had retreated to its natural position under the false ribs.

After the operation, which, as the woman herself stated was not at all painful, the pulse, formerly weak and irregular, regained its power and regularity. The patient ceased to complain of a feeling of extreme weakness which had prevented her from sitting up through fear of syncope.

All the effused fluid, however, had not been evacuated: there was still dulness as high up as the nipple, but respiration was heard in every part of the chest. At a point where a few minutes previously

no sound was audible, we now heard blowing, vocal resonance, and egophony.

The absorption of the fluid took place gradually, and during some following days nothing noteworthy occurred. The general condition of the patient went on improving: by the 15th May, resolution of the pleurisy was complete, and recovery was sufficiently perfect to allow her to leave the hospital at her own request. However, on percussing the chest, I still found dulness, or a hardness of sound from the infra-spinous fossa down to the lower part of the chest. This dulness remains a long time after the most ordinary pleurisy, being caused by the presence of false membranes which require a certain time for absorption. On auscultation, I perceived the vesicular murmur everywhere, but it was accompanied by coarse and subcrepitant mucous râles.

Gentlemen, similar cases will no doubt occasionally come under our observation: but I could not allow this opportunity to pass without speaking to you of paracentesis of the chest in the consecutive effusion of pleurisy; and with your permission, I propose to devote several lectures to the development of this grave and important subject.

It will be granted, I trust, that I seldom speak of myself. Indeed, generally, I attach very little value to questions of priority. I am entitled therefore, once, in passing, to lay claim to as much as belongs to me, in respect of paracentesis of the chest. I make no pretension to being the originator of the practice: I have not invented an instrument for the more easy performance of the operation, nor have I recommended any operative proceeding which was not previously well known: but I conceive that, if not the first, I was at least among the first to point out in a precise manner the necessity of resorting to paracentesis in pleurisies followed by a great amount of effusion. I established with precision—perhaps with more precision than had been previously attained—the indications for operating; and I believe I popularised the method which is now generally adopted, thus entitling me to be looked upon as having somewhat contributed to the progress of the therapeutics of pleurisy.

Let me tell you, Gentlemen, how I was led to inculcate the necessity of surgically interposing in the treatment of extensive hydrothorax. In 1832, a woman aged 50 was admitted to the Hôtel-Dieu, and became a patient in these wards, of which I then

had charge jointly with Dr. Réeamier. For five days, she had been suffering from acute pleurisy. The breathing was exeeedingly oppressed: on the left side, there was eomplete dulness: the heart was pushed over to the right side: the ribs were far apart. A large blister was applied to the ehest: digitalis was administered: in a word, active treatment was instituted. The woman died on the day following that on whieh she was admitted to the hospital. At the autopsy, we found the left pleura distended by an enormous quantity of limpid serum, in whieh fibrinous flakes were floating. The lung was squeezed up against the vertebral column; and both the pulmonary and eostal portions of the pleura were slightly eoated with false membrane. We found no tubereular produets, nor any other serious lesion.

This ease was a direet contradietion to what I then believed, in common with the majority of authors, as to the small degree of danger attaching to an attaek of pleurisy: more extended experience has convineed me how erroneous were the ideas entertained on that point. Other unfortunate eases observed by me and others have negatived the law laid down by Dr. Louis, adopted by his pupils, and re-eehoed by numerous physieians, to the effeet, that pleurisy is never an immediate eause of death—a law, be it noted, founded on a series of 150 eases of simple pleurisy whieh terminated in reeovery.

One of my pupils, Dr. Laeaze du Thiers,[1] has eolleeted a number of eases, some communicated by me and others derived from different sourees, whieh absolutely demonstrate that, notwithstanding the famous law of Louis, it is possible to die, and to die suddenly, from acute pleuritie effusion. Very reeently, my friend Dr. Lasègue saw a young physieian die from this eause, at the very moment that he was about to make the puneture.

On 7th April, 1843, I reeeived into bed 31 of St. Anne's ward of the Neeker Hospital a woman of 42 years of age, with paralysis of the inferior extremities, bladder, and rectum. The intelleetual faculties were unimpaired. The paralysis, which did not affect the superior extremities, had set in suddenly three years previously, and had not sinee that time beeome modified.

Ten days before admission, this woman was seized with stiteh in

[1] Lacaze du Thiers :—De la Paracentèse de la Poitrine et des Épanchements Pleurétiques qui nécessitent son Emploi. [*Thèse de Paris*, 1851.]

the side, cough, dyspnœa, and fever. On examining the chest, I at once detected pleurisy, with effusion on the right side. Dulness extended up to just below the clavicle: egophonic resonance of the voice and bronchial blowing were audible. The cough was dry. Some relief followed a bleeding which I had ordered, but the oppression of breathing continued very urgent. Next day, the orthopnœa assumed very great intensity. The pulse became small and wretched; and finally, death took place, without a struggle, twelve days from the beginning of the disease.

I shall say nothing of the lesions of the nervous system, which were not at all of a severe character. In the right pleura, there was an enormous effusion of purely serous character: the lung, pressed against the vertebral column, was shrivelled and covered with soft false membrane, which had a reticulated appearance: some fibrinous flakes were floating in the effused serosity.

The occurrence of this case of sudden death, from acute pleurisy with profuse effusion, recalled to my recollection that which I had observed eleven years previously in the wards of Dr. Récamier. It set me to think. I asked myself, whether the fatal issue might not have been prevented in both these cases by rapidly disembarrassing the chest of the fluid which it contained, and to the presence of which the untoward symptoms were due. I asked myself, whether under such circumstances paracentesis was not distinctly indicated.

In the same year, and at an interval of exactly a month—on 8th May, 1843—a seamstress, thirty years of age, was admitted to St. Theresa's ward, bed 8, for pleurisy without effusion: she too was carried off in consequence of paracentesis not having been resorted to. This woman had been delivered at the Maternity Hospital on the 19th of April, and had left that institution, in good health, on the 27th, except that she had a slight cough, which had existed for four days. Next day, the 28th, she fell ill, in consequence, perhaps, of the imprudences to which these unfortunate puerperal women so frequently expose themselves. On the same day, the 28th, she had fever, slight oppression, and an increase of cough. These symptoms increased up to the 8th May, when, along with her infant, she was admitted to my ward for nursing women in the Necker Hospital. The lochial discharge, which had ceased for some days after the fever set in, was not long in re-appearing, and at the date of admission, was flowing in a normal manner: the secretion of milk was scanty.

On the 9th May, the twelfth day of the disease, at my visit in the morning, I dictated the following report:—"Oppression, without orthopnœa: countenance somewhat anxious: a dry but not frequent cough: expectoration frothy and scanty, and having the appearance of saliva. Complete dulness on the left side of the chest as high up as the lower margin of the clavicle: a considerably arched appearance of the chest in front: respiratory sound absent, but there can, however, be heard a distant, very feeble murmur, without egophony and without vocal resonance. On the right side, respiration is puerile." The heart was noted as being beyond the median line. I ordered four basins of blood [*quatre palettes*][1] to be taken from the arm, and put her on strictly low diet, broth only being allowed. She was recommended to drink very little.

Next day, there was no change in the condition of the patient. It was observed, that the blood drawn on the previous evening was very much cupped. I ordered the treatment to be continued.

Between the 11th and 17th May, there was a slight amelioration, which, however, was soon succeeded by an exacerbation of the malady. There was a tendency to syncope. Two flying blisters were applied to the affected side, at the interval of a few days; and diuretics were administered.

On the 17th, the state of the patient was manifestly worse. She lay on her back, without pillows, and did not seem to have any oppression: nevertheless, her countenance was pale and anxious, and her eyes wide open. Her respiration was feeble and imperfect: her pulse was miserable: her intellectual faculties, however, were unaffected. It seemed as if she were dying, suffocated by a power against which she had ceased to struggle.

The two cases which I have just related to you presented themselves to my mind: I saw that my patient was in similar peril, and paracentesis suggested itself. But as it was a somewhat unusual proceeding to resort to this operation at the twentieth day of a pleuritic attack, as the operation was at that time loudly condemned in acute cases of effusion by all French physicians, and indeed in all cases of hydrothorax; as the oppression of the breathing did not seem to me to be very great, I yielded to the culpable weakness of preferring to wait: in fact, I wished to avoid the imputation of rash-

[1] A "*palette*" is a basin used for receiving blood taken by venesection. It contains 75 grammes, *i.e.* 8½ fl. ounces.—TRANSLATOR.

ness. I directed my *interne* to watch the patient, and to puncture the chest in accordance with a plan we agreed upon, should life seem in extreme jeopardy. My pupil saw the patient at seven in the evening, for the last time. She did not then appear to him to be in a worse state than in the morning: believing that paracentesis might be delayed, he left her for a little. Within an hour, the unfortunate woman died without a struggle.

At the autopsy, we found that the heart was pushed quite to the right side, and that the left pleura was distended by an enormous quantity of fluid, which we estimated at not less than four litres. This serosity was limpid in the upper part of the chest, but in the depending parts it was sero-purulent: the lung was shrivelled, and was squeezed up against the vertebral column. At the summit, it was closely adherent to the costal pleura, and at that point there was a cicatrix consequent upon softened tubercle. In no other situation, did we find any appreciable organic alteration.

I was, as you may suppose, shocked by the death of this woman; and, too late for her, I perceived the necessity of having recourse to paracentesis at the earliest possible opportunity in similar cases. I had not long to wait for an occasion to give practical effect to this conviction.

In the following September I went to Tours to see my mother, who was dangerously ill. During my absence, I had been sent for by my excellent friend, M. Michel Masson, the dramatic author with whose name you are familiar, to see his daughter. She was a young lady of sixteen, who generally enjoyed good health, excepting that she had great nervous irritability. During the ten years that I had been the family physician I had hardly once been consulted regarding her.

On Sunday, September 3rd, 1843, she had fever and loss of appetite. On the 5th, she took to her bed. I did not see her till the 8th. I then observed that the skin was very pale, and that there was considerable fever: she had a little dyspnœa, but neither cough nor expectoration: there were no symptoms of any gastric affection. On exploring the chest, I found that there was enormous effusion in the left pleura, ascending as high as the clavicle. Everywhere complete dulness existed; and in no situation could I hear the respiratory murmur, blowing, or œgophony. The heart was twisted to the right, and occupied the median line. I bled her

from the arm, prescribed calomel, and recommended her to drink sparingly.

On Monday, September 11th, the eighth day of the attack, there was a great increase in the severity of the symptoms. The skin was cold, and the face pale. On account of the orthopnœa, the young lady was obliged to sit up in bed supported by pillows. She had a tendency to syncope, and groaned without ceasing. I applied a large blister to the posterior aspect of the chest.

I resolved to perform paracentesis; and as the indication to operate was urgent, I did not wish to have a consultation, fearing on the one hand, that a meeting of doctors upon her case might alarm the patient, and on the other, that the conflict of opinions, certain to arise, might lead the family to fatal indecision. Consequently, when I arrived on the Tuesday morning—the ninth day of the illness—I was provided with the necessary instruments, and perfectly determined to fulfil the commands of duty, without making the slightest parade, just as if I were about to do the simplest thing imaginable. I found the patient at the brink of death; and reproached myself for having on the previous evening postponed a proceeding which had become so peremptory as to admit of no delay whatever.

I performed the operation in a way which I shall point out to you, when I come to speak of the manual operation of paracentesis, which is a very simple affair. I withdrew about 800 grammes [between one and two pints] of transparent serosity, having a beautiful amber colour. It retained its transparency till next day, but there was visible in it, a sort of soft, shreddy tissue, evidently formed of fibrine condensed by the cold. Although I could easily have withdrawn a larger quantity of the fluid, I did not wish to do so, being satisfied with having removed the excess which rendered the effusion mortal, so reducing the case to simple pleurisy, curable by ordinary means.

When the operation was completed, the young lady seemed to return to life: she breathed easily, had no longer an anxious countenance, and her pulse had regained some degree of volume. The lungs and heart had returned to nearly their normal position: the respiratory murmur was once more audible: there was a little tympanitic resonance in front, at the upper part of the chest, which, at the time, I thought was attributable to the entrance of some bubbles of air during the operation. I was not then acquainted

with the exaggerated resonance observable in the majority of pleuritic cases—that peculiar resonance which, at a later date, Skoda discovered and made known. During the night which followed the operation, the patient slept six hours.

Next morning, she was in a very nervous state, but respiration was easy: she spoke fluently, and made use of long phrases without taking breath. Her countenance was calm. The skin was rather hot, and the pulse 112.

On the second night after the operation, she slept eleven hours. There was a notable diminution in the quantity of effusion in the chest: the heart was more and more resuming its place on the left, although it was still beyond the median line. Anteriorly, the dulness did not rise higher than the fourth rib.

The subsequent history, I shall rapidly sum up. Under the influence of flying blisters applied to the chest, diuretic drinks, and digitalis, improvement went on rapidly. On 28th September, sixteen days after the operation, the pulse was 80: she had a decided relish for her food: the menstrual flux came at its proper time, but not in normal abundance. From that time, all the morbid symptoms disappeared: respiration became natural, and the patient entered upon a convalescence which had a favourable issue.

This case taught me a great lesson. It prevented me from having any hesitation in acting in the same way under similar circumstances. Having had three similar cases of success, I hastened to publish them. They formed the subject of a memoir which was read to the Academy of Medicine in 1843: during the following year, I communicated a second memoir to the Academy on the same subject.[1] In 1846, the duty of reporting on these two memoirs was entrusted to my honourable colleague Dr. Bricheteau, in whose presence I performed paracentesis of the chest upon a girl of fourteen years of age, who made a rapid recovery. In the report of this excellent practitioner, which is a real masterpiece of erudition and criticism, the conclusions drawn by me are adopted with very slight modifications. The report hardly gave rise to any objections, so that the discussion upon it attracted no attention.

I now more than ever followed out my views in this matter: successes were multiplying, and encouraging me to pursue the same

[1] *Bulletin de l'Académie de Médecine*, T. ix, p. 138; Ibid., T. x, p. 517.

course, when, in 1850, having asked my colleagues of the Medical Society of the Hospitals to give me their opinion upon a case of death following paracentesis of the chest, and information upon a peculiarity in the case, I was led to explain my views on the general question of paracentesis. The controversy was animated; and I found myself confronted by as violent an opposition as that which I encountered when I published my first cases of tracheotomy.

Ill-natured insinuations were not wanting upon that occasion. When I brought forward cases of children cured by tracheotomy, I was told that they had never had croup; that only those children had had croup whom I had failed to save: I was even calumniated. According to my custom, I made no reply. Influenced only by an approving conscience, I continued to pursue the practice, hoping that truth would, sooner or later, carry the day. In respect of tracheotomy, I attained my object; for that operation has for a long time been generally acknowledged as indicated in the circumstances in which I declared it to be necessary. Paracentesis of the chest was not met by the same objections, but, nevertheless, encountered many opponents. When, however, it became known that I had operated in from fifteen to twenty cases, in two instances among others upon Parisian physicians, without having had one failure to deplore; when many of my fellow practitioners in the hospitals and in the town, (my youthful colleagues be it observed, for the elders hardly like to follow the example of juniors), when, I say, many of my colleagues had also operated successfully, paracentesis was proclaimed as a good means of treating acute pleurisies accompanied by profuse effusion, just as tracheotomy had ultimately been accepted in the treatment of croup.

Far be it from me, Gentlemen, to appropriate to myself the honour of the discovery. Paracentesis of the chest has been practised from very remote times; but, exposed to varying fortunes like every other therapeutic measure, it was all but abandoned, or at least was reserved for exceptional cases. It was resorted to distrustfully, and then only when imminent peril justified extreme daring. If it have now taken that place which it ought always to maintain, if it be now mentioned among modes of treatment which have the advantage of being free from danger, I conceive that I have contributed to bring about this change of opinion by my works, by the indications for operating which I have furnished, and in a special manner, let me add, by the success which has attended my practice.

Before I point out to you the circumstances in which this method of surgical interference is indicated, allow me briefly to recapitulate the history of paracentesis of the chest, following it in succession through its different phases. I cannot explain to you in any other way why an operation, never before adopted as an ordinary practice, is at present performed in all places, and by all practitioners.

The earliest data relating to puncture of the chest are as old as the school of Hippocrates. The operative procedure indicated in his times is followed in the present day. There are two modes of operating: we either open into one of the intercostal spaces, or we perforate a rib. The intercostal space may be opened by actual cautery or bistoury. Whatever method is selected, it is essential that the wound remain open till the fluid is entirely evacuated, and till there is no tendency to more effusion. If the opening should have a tendency to close, this must be prevented by the introduction of a metallic sound.

Such is the operative basis upon which surgeons have proceeded since the time of Hippocrates; and it is not uninteresting to see how little they have added to the old traditions. Galen simply repeated the rules laid down by Hippocrates: Celsus did not describe the operation very happily; and paracentesis almost forgotten by the writer who for so long a period was the sole authority on the subject, fell into discredit among the Greeks and Romans, and was not revived by the Arabs.

During the middle ages, it was discussed, whether it were better to make the opening into the chest by steel or fire; but it was scarcely admitted that there were any cases except those of surgical lesions in which paracentesis ought to be performed.

About the sixteenth century, trepanning the ribs was revived, after having been almost abandoned. About the same time, detersive injections, which had been recommended by Galen and Rhases, were again advocated as a necessary element in the treatment. We find, however, that in those times, the operation was seldom tried, and that, even in extreme cases, it was generally rejected by the greatest surgeons. Fabricius of Acquapendente, for example, regretted that it had fallen into desuetude. A more attentive study of facts, however, and a less servile obedience to tradition, led to some important observations.

It was remarked that in penetrating wounds of the chest, recovery seemed to take place most quickly, when the wound was closed

early: and it was then asked, whether it would not be expedient to close the opening made in paracentesis for empyema, in place of leaving it patent for an indefinite period.

From the seventeenth to the eighteenth century, the operation of paracentesis was the topic of numerous surgical treatises. In 1658, Bontius for the first time took up in a precise manner the subject of the introduction of air into the chest, which till then had been hinted at, rather than discussed. He declared that there was nothing to dread in the contact of air, and considered injections as a perfectly sufficient means of combating the consequences which were dreaded. Bartholin maintained the opposite opinion: he insisted that the opening ought to be closed with the least possible delay, to prevent at all hazards contact with the air. The indication for the operation was then laid down; but two centuries elapsed before a satisfactory manner of fulfilling it was attained.

The more that attention was directed to the question of the admission of air, the more was the manner of operating modified. The fluid was evacuated by aspiration and suction: this practice was at first very timidly pursued, in accordance with Scultet's example; but it became afterwards in vogue with the masters of surgical art.

Surgeons had now entered upon a new and very propitious path. In place of discussing theoretical probabilities, they proceeded to direct examination of facts. As cases of paracentesis were still of rare occurrence, they based their observations on examination of wounds of the chest. They thus, from considering the small number of untoward occurrences which such accidents lead to, became less distrustful of puncturing the chest, and were even led to believe in the harmlessness of the operation.

As a consequence of this tendency, felt rather than expressed, physicians began to study the question of puncturing the chest in hydrothorax. In 1624, Gerome Goulu alleged that he succeeded more frequently in hydrothorax than in abdominal paracentesis in ascites. Twenty years later, we read in the writings of Zacutus Lusitanus that paracentesis was as much indicated in cases of serous effusion into the chest as in cases of empyema, if puncture was the only means of evacuating the fluid. Some time afterwards, this practice was put in force by Willis; Lower also mentions a case; and subsequent authors quoted these cases as an encouragement to the performance of paracentesis of the chest for the removal of serous effusion.

About the middle of the eighteenth century, when perforation by

the actual cautery, which till then had its partisans, was abandoned, it was proposed to substitute for the bistoury, a trocar such as was employed in abdominal paracentesis. In 1765, Lurde recommended the use of this instrument, which, however, had been proposed nearly a century before by Drouin. Lurde's recommendation was timidly given, through the fear which he had, that in using the trocar there was a risk of wounding the lung. He advised the operator to close the canula with the finger at each inspiration, leaving it open during expiration, so that the entrance of air might be prevented. Lurde's recommendation to employ the trocar was not so well received as might be supposed; and among its adversaries were Chopart and Desault. The use of the trocar was alleged to be a coarse mode of proceeding, and to involve the risk of wounding the intercostal artery and the lung. More than a hundred years elapsed before the suggestion of Drouin found support; and we can still see with what difficulty it obtained adherents, notwithstanding the future which was in store for it.

Finally, in 1808, Audouard raised anew a question, considered as definitely settled, attacking a universally admitted conclusion. From the time that paracentesis was first practised, the rule laid down was to allow the fluid to flow only little by little. On the first day, a portion was drawn off, on each succeeding day, the evacuation of a small additional quantity was promoted. This precept originated in the conviction, that if the fluid escaped suddenly, the result would be a vacuum in the chest causing the death of the patient. This hypothesis, afterwards admitted to be unsatisfactory, was replaced by another explanation; but the rule remained. Audouard maintained and proved that even the sudden evacuation of the fluid is not attended by the drawbacks which had been supposed to exist.

Between 1808 and 1843, when I published my first work on the subject, the manner of performing the operation had scarcely undergone any modifications, suggested changes not having been sanctioned by experience. Certain facts, however, had been ascertained: it had been found that the fluid might be evacuated rapidly, and at one time; also, that while it was incumbent on the operator to prevent as much as possible the entrance of air, there need not be much alarm as to the deleterious consequences of such an occurrence. The soundness of these two principles being admitted, it followed that the trocar was considered preferable to the bistoury; that perforation of the ribs ceased to be practised; and that various plugs were invented as

substitutes for the finger, which Lurde recommended to be placed at the external orifice of the canula.

Every apparatus constructed for adaptation to the trocar, with the view of preventing the introduction of air and leaving free passage to the fluid, was made on the principle announced almost simultaneously by Schuh and Reybarb, but which has been especially elucidated by the former. The apparatus of Schuh, a complex machine composed of a system of plugs and reservoirs difficult to work, has been abandoned by its inventor. The simplicity of the trocar devised by Reybarb has led to its adoption by all operators. Its peculiarity consists, as you know, Gentlemen, in arming the free extremity of the canula with a piece of goldbeater's skin, which being rolled round the instrument, is then softened by water. The goldbeater's skin thus adjusted, acts as a plug, and has the advantage over every other mechanical contrivance of requiring neither precision nor repairs.[1]

In summing up what has been said regarding the operative proceeding itself, you perceive that a certain number of desiderata have been pointed out; and that all of them have now been so well supplied, that there is not much scope for future improvement. You perceive from what has been said, that even at the time when paracentesis of the chest was performed under conditions and with instruments far from favorable, it took its place among the least delicate operations of surgery.

Nevertheless, puncturing the chest continued to be looked upon as one of those bold measures only sanctioned by the existence of danger of a certain urgency. The cause of this timidity arose from insufficient knowledge of the indications for operating, and not from any imperfection in the mode of performing the operation.

It is impossible for any therapeutical means to be regarded with general favour, be it a medicine or a surgical operation, unless it be suited to a determinate exigency. A remedy will remain unemployed, so long as the circumstances which demand its employment are imperfectly known. Or at most, an experimentalist tries it at a time, in a sort of hap-hazard way, and announces a successful result: others, who attempt to follow in the same direction, either use the remedy too timidly or too indiscriminately, thus discrediting it, and causing it to fall into oblivion. Such has been the fate of

[1] Gaujot:—Arsenal de la Chirurgie Contemporaine. Paris, 1867.

paracentesis of the chest. It is not surprising that de Haen should have asked in reference to hydrothorax:—"*Cur ita laudata paracentesis sive ut primum sive saltem ut alterum adhibendum auxilium spatio XXIII seculorum theoretice commendetur et vix unquam instituta legatur?*"

I have now rapidly sketched the history of the operation; but to make this history practically instructive to you, I must state the indications for resorting to paracentesis.

When Hippocrates recommended tapping of the chest, he distinctly indicated the intention of the operation—the evacuation of fluid contained in the thoracic cavity; but he did not furnish signs sufficiently precise to enable effusion to be detected during life. His description of symptoms is applicable to affections of very different kinds: it comprises, along with dropsy of the chest, hemorrhagic and purulent effusions, and in particular hydro-pneumothorax. Thus, the beautiful experiment of succussion, which still holds its place among the most valuable physical signs, is given as an absolute criterion. It had been perceived that the local phenomena were deficient in precision, and by way of supplementing them, a number of general phenomena, still more deceptive, were added. It is curious to see, that at the commencement of the nineteenth century, the diagnosis of effusion into the pleura had not gained anything in respect of exactitude. The hippocratic description, invariably reproduced by all succeeding authors, is repeated by Mursinna, who, however, in 1811, had operated four times, and twice successfully, guided only by these untrustworthy data. In his remarkable dissertation, the German surgeon insisted that the sound caused by succussion was an indispensable element in the diagnosis. He speaks of œdema of the inferior extremities and scrotum as a symptom almost invariably present; and adds, without making more direct reference to the discovery of his fellow countryman Avenbrügger, a statement to the effect, that a dull sound of a very peculiar character is sometimes heard on percussion. No one has shown more clearly than de Haen, the inadequacy of the diagnostics. His monograph on hydrothorax is in fact an elaborate criticism upon the phenomena which his predecessors had indicated as characteristic:—"*Suspicio morbi duntaxat est eaque cum aliorum morbis signis ita intricata, ut certi quid concludi nequeat.*" He elsewhere insists upon the deceptions which beset physicians:—"*Subdolus hic morbus raro dum curabilis est cognoscitur.*"

The result of this diagnostic uncertainty, which was felt and proclaimed by all good observers, was the restriction of paracentesis of the chest to cases extremely limited in number, and belonging to the domain of surgery. It was thought necessary to wait till the chest was enormously distended, till an intercostal space was visibly elevated by the tension caused by the pus seeking to make an exit for itself; and, generally speaking, the rule was to operate only in traumatic cases.

Nevertheless, in spite of so many reasons for abstaining from operating, and though there was very little actual experience to appeal to, there was not wanting on the part of physicians belief in favorable results, and the hope of success. They asked themselves the reason for delaying the operation to so advanced a stage of the case: they were convinced by reasoning rather than by facts—but they were convinced. This remark is applicable to more than one writer. So far back as 1624, Goulu, as I have already stated, alleged that paracentesis of the chest in hydrothorax was more frequently successful than paracentesis of the abdomen in ascites:—"*Ergo in thoracis quam in abdominis hydrope, paracentesis tutior.*" Such is the conclusion of his dissertation. In 1774, Majault took up the same thesis, and thus summed up his views:—"*Ergo hydropi pectoris paracentesis.*" De Haen, too, had said:—"*Ut si hydrops pectoris cognoscatur mature, nil est paracentesi tutius.*"

The great discovery of Laennec changed the aspect of the question: in the room of confused and inextricable symptoms, auscultation substituted the simple and positive elements of diagnosis. Effusions into the chest were henceforth considered to be among the diseases which were most accessible to the investigation of the physician. From that moment—the obscurity being apparently dispelled—the desire so often expressed of extending the practice of paracentesis of the chest seemed about to be realised. It, however, happened otherwise. Laennec, with his usual sagacity, had defined all the indications of the operation. He recommended its performance in cases of acute pleurisy with effusion, in which the effusion, from the first very profuse, increases so rapidly, that at the end of some days it produces a general or local condition of great seriousness, possibly threatening suffocation, and constituting what has received the name of *acute* empyema. He also recommended it as a last resource in chronic pleurisy, when every means had failed to promote absorption of the fluid; but he added:—"The operation for

empyema is seldom successful." That depends on several causes, all of which have not been equally well appreciated. With the exception of particular conditions of organs contraindicating paracentesis, the condition which Laennec considered as most opposed to success was flattening and loss of elasticity of the lung, from its being covered by false membrane. Hence, he thought that there is a better chance of success in acute than in chronic empyema. The usual mode of operating did not appear to him to be susceptible of much improvement; and though he dwelt upon the danger resulting from the introduction of air, he does not seem to have inquired into the means of preventing this occurrence. "Puncture with the trocar," says Laennec, "in an intercostal space, was several times tried: Morand among others had recourse to it several times. My friend Professor Récamier employed it several times, making use of a very small trocar. I have myself often had recourse to it, but have never obtained by it permanent success." Three pages farther on, Laennec expresses the conviction that "the operation for empyema will become much more common and much more useful, in proportion to the diffusion of the employment of mediate auscultation."

As you perceive, Gentlemen, Laennec does not by any means enunciate his opinion in a decided manner: he raises with one hand that which he pulls down with the other. While he promises fortunate results from the diagnostic means with which he endowed science, he speaks doubtingly of the successful results of paracentesis. He only cites unsuccessful cases, and lays stress on the anatomical conditions, only that he may bring out into stronger relief those which appeared to him to be the most unfavorable. Distrust, though tempered by the expression of better hopes for the future, was too distinctly shown by Laennec to encourage new attempts. What was the consequence? When ten years later, in 1835, the question of paracentesis was brought before the Academy of Medicine, upon the occasion of Dr. Faure's memoir being read, the discussion was confused and misty, opinions of a contradictory character being stated: on all sides, proofs were wanting; and prolonged debates terminated without any conclusion being arrived at.[1]

There was a disposition to follow the authority of Laennec in France, the country in which auscultation was discovered, and an inclination

[1] *Bulletin de l'Académie de Médecine*, 1838. T. i, p. 62.

to dispute the efficacy of paracentesis in the different forms of effusion into the chest which physicians are called upon to treat; and surgeons continued to employ it only in traumatic effusion. But in foreign countries, the new means of diagnosis excited better hopes from the operation.

In 1834, Becker published, at Berlin, a monograph on chronic pleurisy, in which, after having explained how the progress of thoracic examination had enabled the operation to be better applied, he detailed five cases of chronic pleurisy in which paracentesis was performed at the request of Dieffenbach; and upon his title-page, he placed these words—"*Melius est anceps remedium quam nullum.*" In 1835, Thomas Davies set himself after a somewhat awkward fashion to refute the opinion of Laennec, whom he represented as utterly denying the utility of the operation. In opposition to the opinion of the French physician, he declared that paracentesis is useless in pneumothorax, but that it renders very marked benefits in cases of hydrothorax and empyema, particularly in children. His plan was to make the puncture in the intercostal space with a small trocar, without using any accessory apparatus to prevent the entrance of air. He recommended—and the recommendation has been long followed in England—that the operation should be preceded by an exploratory puncture with a needle, to ascertain the nature of the effused fluid and its degree of consistence, as well as other points such as the presence or absence of false membranes.

The support given by Thomas Davies to an operation then so little in favour was not without a good effect. His opinion was ignored in France, but had many advocates in his own country, where Davies became an authority with all who made new trials of the operation: the feeling in its favour, however, was not a general conviction. While some performed the operation, and lauded its results, there were others who abstained from having recourse to it, and were not remiss in arguing against it. Stokes,[1] and afterwards Watson, insisted upon the evils resulting from paracentesis, which they alleged converted a serous into a suppurative inflammation—a false doctrine which has been recently revived. Holding these views, they maintained that the operation ought not to be performed, unless the life of the patient was in imminent peril.

In Germany, however, confidence was sustained. Schuh of Vienna gave in his adhesion to the doctrines of Becker. In 1839, Schuh in

[1] Stokes :—Diseases of the Chest: Dublin.

his "Dissertation on the Influence which Auscultation and Percussion are entitled to exercise on Practical Surgery," declares that paracentesis is a radical cure in cases of chronic thoracic effusion, whether the effusion has or has not followed an acute attack. In that work, he lays down the principles which three years later he applied in practice.

Such, Gentlemen, was the state of the controversy upon this question in therapeutics, when I contributed my own researches to its elucidation. Contradictory opinions were maintained with equal keenness on both sides. Dr. Reybard had already published an account of his ingenious instrument;[1] but being more occupied with the mode of operating, than with the indications for operating, he added nothing to what had already been taught on the subject. He admits that he had had few opportunities of personally applying his method in practice, and from want of cases to appeal to, he supposed that dropsical effusions into the chest, generally caused by an inflammation of the pleura, ought to be cured by a single tapping. The remainder of his exceedingly interesting dissertation is specially devoted to wounds of the chest.

Towards the end of 1841, two professors of the Vienna School of Medicine, Schuh[2] and Skoda published an important work on this therapeutical subject. As I formerly mentioned, Schuh had already, on theoretical grounds, lauded the good effects of paracentesis of the chest. Their monograph, which has become a classical work in Germany, deserves to occupy a distinguished place in the history of paracentesis of the chest. They begin by laying down the principle that recovery from pleurisy generally takes place when the effusion is not excessive in quantity, and when the case is without complications. Even when the effusion is profuse, they say that nature will, when aided by known means, accomplish a cure; but that the time required will extend to months, or it may be to years. The evils which follow pleurisy are deformity of the chest, anæmia and its worst consequences, viz. syncope, sudden death, formation of tubercle, and hypertrophy with dilatation of the right side of the heart.

[1] Reybard:—Mémoire sur Les Epanchements dans la Poitrine, et sur un Nouveau Procédé Opératoire pour retirer les fluides épanchés sans laisser, pénétrer l'air extérieur dans le thorax. [*Gazette Médicale*, for January, 1841.]

[2] Schuh:—*Medicinische Jahrbücher der k. k. Osterreich. Staates.* Wien: 1841.

If the effusion be to the extent of several pounds, and the fever have ceased, paracentesis is indicated, provided there has been no amelioration of symptoms for three weeks: the operation will prove either a radical cure or a palliative.

Both authors set themselves to refute the arguments adduced against paracentesis, and to explain in detail the different stages of the operation. They recommended that the whole of the fluid should not be withdrawn at once, being afraid that the sudden expansion of the lung, and replacement of organs pushed out of place by the effusion, might be productive of injurious consequences. This fear is chimerical, as I shall show you.

The work of Schuh, though supported by the authority of Skoda, did not, even in Germany, meet with the approbation which it deserved. In foreign countries, it remained unknown; and I have not once seen it quoted by French or English experimenters.

Almost about the same time, just as if in the history of this operation, it was fated that every hope of success should be counterbalanced and contradicted, Hope dictated his "Notes on the Treatment of Chronic Pleurisy," which were published in the *Medico-Chirurgical Review* (of London), in 1841. He therein endeavoured to prove that pleuritic effusions did not require the aid of surgery, and that the resources of the materia medica were always sufficient for their treatment. Concurring with the physicians, who, upon the occasion of the discussion before the Academy of Medicine, maintained that pleurisy is never fatal, concurring also with Stokes and Watson, Hope declared that the want of success in the treatment arose from the timidity with which the remedies were administered. He spoke very strongly in favour of the plan of pushing resolutely mercurial medicines, and giving a sustaining diet embracing animal food, even when there existed fever; and he recommended certain diuretics. He details thirty-three cases of chronic pleurisy cured by this plan of treatment.

The discussions excited by these writings were not very numerous, and were soon forgotten. Attention was not fixed upon the indications, upon the advantages or danger of paracentesis; and what appeared at that date in the medical periodicals was only an occasional report of cases in which the operation was performed, both the nature of the cases and the results being very various.

In 1844, my memoir read before the Academy, by awakening a fresh interest in the subject, called forth new researches. In

England, a similar impulse was given to inquiry. It now began to be felt, that there was a sufficiently solid basis for inquiry, and that consequently, leaving aside hypothesis, facts might be appealed to. In this spirit were conceived two monographs, which close the list of English publications of any importance upon tapping the chest. I refer to the work of Hamilton Roe "On Paracentesis of the Chest in Empyema and Inflammatory Hydrothorax,"[1] and to the writings of Hughes.[2] Roe laid down excellent precepts, and considered the results of experience as very satisfactory. He was not afraid of syncope, which according to the views of objectors, threatened the life of the patient immediately after the operation; nor was he afraid of the entrance of air, against which he took no manner of precaution. The mode of operating which he recommended was exceedingly simple: he reduced the operation to a mere puncture in an intercostal space with a trocar of medium size.

Perhaps, Gentlemen, you may have thought that, considering the nature of the instruction which it is my duty to give, I have gone too minutely into this historical sketch of paracentesis of the chest; I wished to go fully into the matter, because I desired to show how this operation, though recognised in the very infancy of medicine, had had difficulty in establishing itself in the domain of therapeutics, and that its doing so at last was in consequence of the indications having been established in that precise form in which they now exist.

I propose in my next lecture to go into the essentially practical parts of the subject.

Circumstances which render Paracentesis of the Chest necessary.—Pleurisy may be fatal.—Profuse Effusion may cause Sudden Death.—It may occasion Death by Asphyxia.—On the other hand, Paracentesis may accomplish an Immediate Cure: when this takes place, the Temperature of the Body at once becomes normal.—The

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[1] Hamilton Roe:—*Transactions of the Medical and Chirurgical Society of London:* 1844.

[2] Hughes:—*Guy's Hospital Reports*, 1844: and *London Medical Gazette*, 1846.

Continuance of the Effusion in the Chest may occasion Hectic Fever.—The Effusion may become Purulent.—Traumatic Pleurisy.—Pleurisy may occasion development of the Tubercular Diathesis.—Latent Pleurisy is a frequent manifestation of this Diathesis, whether the Effusion remain Serous, or become Purulent, as usually occurs.—Paracentesis is also useful when there exists Hydro-pneumothorax.—Cancerous Pleurisy.

GENTLEMEN:—To justify paracentesis of the chest in pleurisy with a great amount of effusion, it is first of all essential to establish, in opposition to the opinion of Dr. Louis, that pleurisy sometimes terminates in death.

The disease may prove fatal from the immediate effect of excessive effusion.

Death may also be, in an indirect manner, the result of pleurisy becoming the starting point of affections under which individuals sink sooner or later. For example, the mere continued existence of effusion, whether the fluid in the cavity of the pleura remain serous or degenerate into pus, will cause continued fever, hectic fever, by which the patients will become exhausted. Let me add, that from the special nature of the inflammation which gives rise to the effusion, the effusion is often purulent from the very first.

Moreover, a persistent determination towards the thoracic viscera is calculated to lead to the development of the tubercular diathesis in predisposed persons.

Finally, the longer a pleurisy continues, the less curable is it, as the lung contracts adhesions in protracted cases which permanently prevent it resuming its place in the thoracic cavity, and discharging its functions.

Let me pass before you in review the different propositions, now generally admitted to be true; and to which general recognition, I have perhaps contributed.

Pleurisy may prove fatal from the quantity of the effusion being very great.

This is a point which I have already established in my last lecture. I established it, not only by appealing to cases which had come under my own personal observation, but also by referring to others which had been observed by physicians altogether reliable, among

whom I may mention Chomel, Brichetcau, and my friend Dr. Pidoux.

Among other additional examples which I could lay before you, let me present to you the following history, all the details of which I have carefully collected.

On 17th August 1847, I was requested to see, in consultation, a man aged 44, who had been ill for six weeks. Up to the date of his attack, he had always had good health. On 3rd July, he was seized with symptoms of inflammatory fever, the result of a chill, commonly called a *coup de froid*. He continued to walk daily from his residence at the Barrière Blanche to the War Office in the rue Saint-Dominique, where he was a clerk. In the evening, on returning from his office, he always complained of fatigue, then of a feeling of oppression, which increased day by day, and which he compared to a military stock [*hausse-col*] painfully compressing the upper part of the chest. On 26th July, he was obliged to give in. The dyspnœa, the feeling of constraint of the chest, and the general debility went on increasing: consequently, he took to his bed, and sent for his medical attendant. At that time, he was coughing a little. The physician who was called in informed me that he found him in a feverish state, with rusty sputa, but without dulness of the chest on percussion. He was bled: but as the bleeding was followed by syncope, the physician was afraid to repeat it. A plan of treatment was then commenced intended to produce revulsion from the lungs to the skin and intestinal canal; while, at the same time, there were administered opiated potions to subdue the cough. It was not till the eighth or tenth day of the attack, that incipient dulness was detected on the left side: the dulness existed throughout the entire left side of the chest, both before and behind. The treatment was then changed to blistering the chest, and administering diuretics. The severity of the symptoms increased: the debility and the fever became more menacing. There was delirium during the night, and profuse sweating which weakened the patient: but the oppression of the breathing was only marked in a moderate degree. As, however, matters were going on from bad to worse, they did me the honour to call me in.

The following is an account of the state in which I found the patient. The pulse was 100, soft, and easily compressed: there was sweating, which was constant, passive, and warm: the skin was rather hot: the face was flushed, the eye expressionless, and

the brain "empty" to use a vulgar phrase. There was no cough, and the respirations were 25 in a minute. The patient was lying in the horizontal position: on my arrival, he sat up in bed, and made no complaint, except that he suffered from fever, and wasting sweats, to use his own expression [*des sueurs qui le minent*]. He only made one request, which was, that the fever might be cut short: he made no mention of oppressed breathing, cough, or stitch in the side. He passed his urine and stools involuntarily. His countenance and appearance suggested the idea of a person suffering from typhoid fever, but with this difference, that the mucous membrane of the mouth was in a normal state.

On examining the chest, I found complete dulness on the left side from summit to base, in front and behind, extending to the subclavicular and infra-spinous fossæ: everywhere, auscultation revealed the absence of all sounds, vocal or respiratory, normal or morbid. On this side, the thoracic walls did not vibrate under the hand when the patient spoke. On the right, there existed normal resonance and supplementary respiration. There was no enlargement of the intercostal spaces, and the chest did not seem to be distended on the affected side. The effusion extended laterally to the middle of the sternum: exactly at this point, and not in its natural position, the pulsation of the heart was felt.

Looking to the great amount of the effusion, and notwithstanding the slightness of the dyspnœa, I was of opinion that any attempt to promote absorption would be useless and even injurious, inasmuch as it would delay the tapping, the only efficacious treatment which could be employed.

However, to enable the patient to wait till the following day, (it was then three in the afternoon,) I prescribed (in place of the strict low diet on which he had been placed) two cups of milk and meat broth, a slightly stimulating potion, and thirty centigrammes of the sulphate of quinine. But at eight o'clock next morning, the patient died, after some hours of dreadful agony, repeated faintings, delirium, but no great amount of dyspnœa. After death, percussion of the chest gave results similar to those of the previous evening.

These unfortunate cases, more of which might be cited, speak too plainly to allow any one to deny that simple pleuritic effusion, may from mere quantity, cause death. We shall afterwards see how this occurs.

Enormous effusion may arise from simple acute pleurisy; and I do

not believe that as yet any clinical observer has discovered signs by which to determine whether an attack of pleurisy is or is not to be followed by extensive effusion. There can, however, be no doubt that Dr. Pidoux made a correct remark when he said, that "very profuse effusion generally supervenes in a peculiar form of pleurisy, quite different from ordinary pleurisy." [1]

It is usual to distinguish two stages in pleurisy. The first is peculiarly the *inflammatory stage*, characterised by anatomical changes described in your text books, and during the progress of which occur in a marked manner the ordinary phenomena of inflammatory fever, with the violent stitch in the side, and the dry pleuritic cough. This stage is of short duration: and some, indeed—among whom is Laennec—have denied that it is at all distinct from the second. The second is the *stage of effusion:* in it, the inflammatory element appears to be relatively feebler, but continues during a period to which it is difficult to assign the limits; and afterwards, the effused products undergo transformations, and originate false membranes.

As an exception to the general rule, the inflammatory element is sometimes very slightly marked—if we judge by the mildness of the local and general symptoms—though the amount of the effusion is considerable.

This is not peculiar to the pleura, being likewise observed in other serous membranes. Observe what takes place in the synovial membranes. In certain forms of articular rheumatism, the phlegmasia, characterised by intensity of local pain and general reaction, is very violent, although the effusion into the joint is inconsiderable: in other cases, the effusion, although proportionate to the intensity of the inflammation, yields as rapidly as the phlegmasia itself: finally, there are cases, unfortunately only too common, in which there is almost no inflammatory action, although there is an enormous synovial exudation, remaining for months, notwithstanding the use of the most energetic therapeutic measures. A similar occurrence is observed in peritonitis, in which the ascitic effusion is never greater than in cases in which the phlegmasia of the serous membrane has seemed to be exceedingly slight. Of this you have recently seen an example in a young woman in St. Bernard's ward, who had hydro-peritonitis for several months.

Acute hydrothorax, that particular form of pleurisy of which I now

[1] Pidoux :—Mémoire sur le Prognostic de la Pleurisie Latente, &c. Paris 1850.

speak, is evidently, in general, associated with a special state of the system, a sort of *serous diathesis*, which may either show itself only by effusion into the pleura, or which may simultaneously manifest itself by effusion into other serous cavities.

Some of you no doubt remember the case of a man, sent to us by Professor Rostan, who died in our wards from double pneumonia complicated with peritonitis. This patient entered the wards of my honorable colleague with extensive pleuritic effusion requiring paracentesis. At the request of Professor Rostan, I performed the operation: a perfectly transparent serous fluid was withdrawn. The effusion having been reproduced, a second operation was performed, when again the fluid was purely serous. Again, there was a return of the effusion. The patient then came into my wards. He had at that time double pleurisy, and also subacute peritonitis accompanied by considerable ascitic effusion.

There was evidently in this individual a peculiar tendency to inflammation of the serous membranes.

At the autopsy, we found the surface of the peritoneum covered with small granulations, which gave it the appearance of the skin of a plucked bird. The granulations had none of the characters of tuberculous products, of which no traces were found in any organ.

Latent pleurisy is a subject on which I have now a word to say.

In consequence of a chill, or, it may be, even without any appreciable determining cause, an individual is seized with rigors, general uneasiness, loss of appetite, pains in the back and limbs, and feverishness: he feels a slight stitch in the side, or perhaps he has no pain in the side: for a few days, he has a short dry cough. These first symptoms continue a very short time, and are so soon forgotten by the patient that he makes no reference to them, unless you recall them to his recollection. The disease has, nevertheless, pursued its slow course: the patient feels that he does not breathe freely, and that he pants on making the least movement: the dyspnœa is so great that he cannot lie on the sound side, or perhaps he finds it impossible to remain in the recumbent position: there is orthopnœa: possibly, the dyspnœa is so slight that the patient is found lying flat on his back, and the oppression of the breathing may be much more appreciable by the physician than by the patient. On examining the chest with the pleximeter and the stethoscope, you discover that there is effusion—sometimes enormous effusion—by which the heart, spleen, and liver are compressed and displaced, by which the chest is deformed in con-

sequence of great distension of the affected side. These extensive effusions lead to very serious dangers.

Sudden death may be one of the consequences. It is not unusual, I repeat, for persons with extensive pleuritic effusion to sink all at once, without having had the breathing much oppressed, at any time, and without ever having had a threatening of suffocation. Death takes place from syncope. In corroboration of this statement, I appeal to the cases, published by my professional brethren, of sudden death occurring under such conditions as I have now described. I could also adduce several similar cases which have occurred in my own practice. The mortal faintings are explained by the great displacement of the heart occasioned by the mass of effused fluid. The heart, as I have told you, is forced out of its natural place: the aorta and large vessels are twisted in such a way as to impede greatly the current of the blood, so that under the influence of an exciting cause, such as the more or less abrupt movement of the body, the circulation is brought to a complete standstill. Perhaps also, death is sometimes induced by the formation of clots in the heart and large vessels, which is liable to occur from the circulation of the blood being impeded.

This opinion, which I announced long ago, has been verified by a case to which Dr. Blachet has directed attention.[1] This physician relates the interesting case of a patient who died suddenly in a faint. At the autopsy, a clot was found occupying the entire extent of the trunk of the pulmonary artery, and, bifurcating, it stretched into the divisions of the third and fourth order of the left branch of the artery. In this case, the pleurisy was chronic: there was about a litre and a half of effused fluid: the heart was not displaced, and the sudden death was probably the result of the blood coagulating in the pulmonary artery.

Although in many cases the patients do not complain of oppression in the breathing, although the existence of dyspnœa is not very apparent to the physician, extensive effusion may nevertheless cause death by asphyxia. In these cases, the asphyxia is slow, the consequence of great embarrassment in respiration, hematosis not taking place in the affected lung, and being but imperfectly performed in the other, from its movements being necessarily disturbed by the liquid which fills the pleural cavities, presses on the mediastinum, and so diminishes the capacity of the other side of the chest.

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[1] Blachet:—*Union Médicale* for February, 1862.

The only way to prevent a fatal termination is the true heroic practice of puncturing the chest and drawing off the effused fluid. This operation is quite free from danger. I shall, however, discuss the objections which have been brought against it, and shall I trust, be able to show you that the imputed drawbacks are purely imaginary.

Besides the case which led to my giving you the present lectures on pleurisy, you have seen many others, in which paracentesis of the chest was practised under similar circumstances, and with equally happy results. The cases go on increasing infinitely; for on all sides, physicians are in haste to publish them. For my own share, I could cite a great many; some of them have occurred in my own practice, and others have been obligingly communicated to me by professional brethren. I shall restrict myself to giving you the particulars of four cases.

The first occurred in my own practice, and is now of old date: the second was reported in the wards of my honorable friend Dr. Horteloup, my colleague in this hospital: the third was communicated to me by my pupil Dr. Bonfils: the fourth you have recently had an opportunity of observing in my wards, and I shall specially call your attention to it, as it presents important peculiarities in respect of the question of temperature.

On Saturday 22nd June 1844, my friend Dr. Patin came before six o'clock in the morning, to take me to see with him Madame Schlaguestad living at 3, rue Marcadet, La Chapelle-Saint-Denis. He had been sent for during the night to this patient, who at the fourteenth day of a pleurisy was so distressed for breath, that her life seemed to be in immediate danger. On Sunday, 9th June, she had felt uneasy, and had had a little pain in the left side of the chest. She had in a casual manner, consulted a physician, who, considering that the breathlessness, pale countenance, and stitch in the side depended on chlorosis, ordered generous diet, good wine, and walking exercise. The unfortunate patient carried out only too scrupulously this fatal prescription; with energy she fought against the fever under which she was sinking: at last, on the eighth day of her pleurisy, conquered by the disease, she took to her bed, and called in Dr. Patin, who had no difficulty in discovering the nature of the case he had to deal with. There was complete dulness on the left side of the chest, from base to summit: the intercostal spaces were distended; and the heart was pushed to the right side.

Active treatment gave temporary relief. On Friday the 21st, she

was in a somewhat improved state, after appearance of the menses : during the evening they ceased. In the night, the dyspnœa having become rapidly worse, and death seeming imminent, Dr. Patin was summoned from his bed.

Dr. Patin found the patient sitting up in bed, supported by pillows ; her face was pale and anxious : her eyes were wide open : her nostrils were in violent motion : and her respiration was extremely difficult. There was complete dulness of the left side of the chest, which was enormously distended : in that situation, a blowing sound and egophony were heard : the cough was moderate. The heart was beating under the cartilages of the right side of the sternum. The pulse was very rapid, and exceedingly weak. The amount of effusion was very great. *Death was imminent.* These were the circumstances under which I was called in. On my arrival, the course to be taken was very soon decided on : paracentesis was immediately performed. By this proceeding, there was drawn off, without much difficulty, exactly two litres by measure [above two quarts]. You can easily understand the extraordinary relief experienced by the evacuation of these sixteen palettes of serosity. The poor woman was in fact restored to life. The heart had resumed its place, and the pulse was full and regular though still somewhat frequent : the dyspnœa was gone. She lay almost in the horizontal position, breathed calmly, and had some desire to sleep. The dulness still continued on the left side of the chest, which now seemed smaller than the right side.

The bronchophony and blowing sound remained as before. I prescribed digitalis, and recommended that the patient should have as little as possible to drink. She had a perfectly quiet day : and during the night, slept seven hours. During some following days, the digitalis was continued, and some purgative medicines were administered.

On Tuesday, 25th, I again saw this lady. The clavicular region and the entire infra-spinous fossa yielded a clear sound, and the respiratory murmur was heard in these situations : from the crest of the scapula to the base of the lung, egophony and bronchophony were audible. The patient had slight fever and a little dry cough, but there was no oppression of the breathing : she had appetite for food. The digitalis was ordered to be continued ; and she was allowed to take light nourishment.

Eight days later, the respiratory murmur was heard as high up as the middle of the infra-spinous fossa : below that point, the sound was

clear: near the angle of the scapula, there was very distinct egophony: lower down, there was a blowing sound and bronchophony: there were no râles: the cough was still dry. Although the appetite was very good, the fever continued. I ordered a blister to be applied to the back, suspended the other treatment, and prescribed a decidedly nutritious diet. Sixteen days after the operation, so great was the patient's restoration of strength, that she was able to walk to the residence of a relation at Clignancourt, a distance of more than two kilometers [more than a mile and a half], remain there during the day, and return on foot in the evening, without being much fatigued. I saw her three days after this imprudent proceeding, when she was very well, had an excellent appetite, and had neither cough nor dyspnœa. Posteriorly, there was still dulness in the lower part of the side which had been the seat of the effusion, but the respiratory murmur was everywhere audible.

The following is the history of the second case. On 5th January 1854, a man, aged thirty-six years, came into Dr. Horteloup's wards in the Hôtel-Dieu. He said that he had been ill for three weeks; but that he had had cough for six months. Upon interrogating him as to his hereditary tendencies and personal antecedents, nothing was elicited indicative of the tuberculous diathesis. It appeared that without any cause appreciable to the patient, he had been seized three days prior to his admission to the Hotel-Dieu with shortness of breath and wandering pains in the chest, but without having any stitch in the side. He had at that time no fever, and was able to continue his ordinary work. In consequence of increased difficulty in breathing, he applied for admission to the hospital.

On admission, he was suffering from extreme anxiety and suffocative symptoms, speaking with difficulty, and in a short, jerking voice. His countenance was pale and blue: the extremities were cold. At the first examination, the chest was evidently distended on the right side. There was complete dulness from summit to base, both before and behind—a little less, however, below the clavicle, and in the upper part of the vertebral hollow. In that situation, and there only, was heard a slight distant blowing sound: the respiratory murmur was everywhere absent. The pulse was quick and small. A large blister was applied to the side; and a bottle of Seidlitz water was prescribed.

There was no change in the state of the patient by the 10th January, except that he felt better. Bear this in mind: it is a

special circumstance, to which I have already directed your attention, and to which I shall have to return. The blueness of the face, especially of the lips, was even more decided. Asphyxia seemed imminent. Under these circumstances, my honourable friend Dr. Horteloup asked me to see the patient. I stated that I considered paracentesis to be urgent; and consequently, it was at once performed by M. Dal Piaz, *interne*. Twenty-two palettes, or in other words 2,500 grammes [more than two quarts and a half] of lemon-coloured serosity, having a somewhat dirty tinge, were drawn off. Immediate relief was experienced. The plessimetric and stethoscopic phenomena were at once modified: there was a diminution in the dulness, and a blowing sound as well as mucous râles were audible. I prescribed the tisane of digitalis. Eight days afterwards, when I saw the patient, he was so much improved in appearance, that I did not at first recognise him. He had regained a plump, healthy appearance. I found him lying on his back.

On 2nd February, he was dismissed from the hospital at his own request. His general state was then very satisfactory. Some harshness of sound remained in the right side of the chest, but the vesicular murmur was heard from base to summit.

The following case, communicated by Dr. Bonfils is not less conclusive than those I have now related to you.

"On the 10th or 11th July, 1861, Madame L., aged fifty-four, residing in the rue Saint-Honoré was seized with serious pulmonary symptoms. The symptoms were of the nature of those which generally accompany the onset of purely inflammatory pleurisies viz. rigors, general pains, burning fever, extreme anxiety, headache, and complete insomnia. The patient complained of an acute pain in the side. Respiration was anxious and panting, the embarrassment being to such a degree as to amount to orthopnœa. When summoned in haste on the morning of the 13th July, the symptoms which I first observed at once directed my attention to the respiratory organs. On examining the chest, I detected, without any difficulty, that there was extensive effusion into the left pleura. The intensity of the fever, and the general constitutional disturbance, showed the serious nature of the case. Blisters, purgatives, diuretics, large doses of the tincture of squills and of digitalis, did not in any degree impede the progress of the effusion, which increased with terrible rapidity."

"On the 18th, the seventh day from the beginning of the attack, the effusion filled the pleural cavity, coming as high up as the

crest of the scapula, and displacing the heart, the apex of which was beating to the right of the median line of the sternum. There was complete dulness throughout the whole of the left side of the chest, where no respiratory sound was heard."

"The tendency to lipothymia being manifest, and syncope threatening, I had no hesitation in proposing paracentesis to the family. My excellent master, Professor Trousseau, kindly gave me his aid on the occasion. The operation was at once performed; and 1,750 grammes [nearly two quarts] of serosity flowed from the canula of the trocar."

"Immediately after the evacuation of the fluid, respiration was heard throughout the whole of the affected side: and percussion gave a resonant sound where before there was absolute dulness. The heart had returned to its place, and the serious symptoms, so threatening before our intervention, had disappeared. The results of the operation were all that could have been desired."

"Next morning, the general state of the patient was satisfactory; and respiration was performed with perfect freedom. I ought, however, to add, that the fluid was reproduced to a small extent; but the daily external application of the tincture of iodine speedily caused it to be absorbed. Recovery was complete on the ninth day after the operation."

This is the case to which I referred. It is interesting, not only because we have, like in many other cases, had the opportunity of clinically observing that convalescence was the direct result of paracentesis of the chest, but especially because examination of the temperature before and after the operation, demonstrated this convalescence materially, it may, in fact, be said, mathematically. The coincidence of the definitive return to the normal temperature with the evacuation of the fluid was at once proof of the cure of the patient, and of the curative effects of the operation.

On 11th June, 1864, I received in St. Agnes's ward a young man aged twenty-three, of very delicate constitution, thin, and pale; but who, nevertheless, did not cough, and who presented none of the rational signs of tuberculisation. Fifteen days before he came into the hospital, he had a slight stitch in the left side, accompanied by a little fever. He had not been confined to bed; and was able to walk to the hospital. Posteriorly, on the right side of the chest, as high up as the spine of the scapula, I found absolute dulness without any blowing sound or egophony: there was also dulness in front.

Within a small space, not larger than a five-franc piece, situated in the upper part of the chest, near the sternum, I perceived skodaic resonance. For twelve days, the state of the patient was almost stationary, the effusion, however, increasing rather than diminishing. From the effusion being on the right side, from the patient being of feeble constitution, and subject to fever every evening, I dreaded pulmonary tuberculisation: and determined to resort to paracentesis. The operation was performed by Dr. Peter, then my *chef de clinique*, on the morning of the 26th day of the attack, the temperature being 38°.7. There was withdrawn from the chest 750 grammes of serosity, which, by pressure in a linen cloth, yielded three grammes and a half [44 grains] of moist fibrin. On the following days, the respiratory murmur was heard throughout the chest, mingled with some subcrepitant râles behind on the right side: in front, in the upper part of the chest, was heard the crackling like that of new leather. On 8th July, the patient left the hospital perfectly recovered.

Let me now tell you what was learned in this case by thermometrical examination. I have already told you that just before the performance of the operation, the temperature was 38°.7: on the evening of the previous day, it had been 38°.6, and for eight days, it had always kept about that in the evening, falling in the morning only from four to six tenths of a degree. On the very evening of the operation, however, in place of rising, as before that date it had usually done, it fell to 38°: next morning, it continued to descend, and fell to *thirty degrees and two tenths*—that is to say, the temperature became normal. In the evening, it fell four tenths, being then 38°.8: and that was the temperature next morning, forty-eight hours after the operation. From that time, till the day on which the patient left the hospital, there was a physiological temperature varying between 37°.6 and 36°.6. As temperature is the best criterion of the febrile state, and as in the case now under consideration, the sudden and permanent fall was coincident with the thoracocentesis, it may be said that the convalescence of the patient began immediately after the operation, and that the recovery dated from the evacuation of the fluid.[1]

In pleurisy, and, in general, in all inflammations of serous membranes,

[1] Duclos:—Quelques Recherches sur l'Etat de la Température dans les Maladies—Thèse (1864); where this case and the thermometrical scale relating to it are given.

thermometrical investigation does not furnish a curve so distinctively characteristic as in certain diseases of regular type, as, for example, in fevers. However, in pleurisy, as well as in peritonitis, it is useful in enabling us at once to distinguish these diseases from certain very painful affections which they simulate. If we find that the temperature remain normal in a patient suffering from intense pain in the side or abdomen, we may, in the former case, conclude that he has pleurodynia and not pleurisy; and in the latter, that the cause of the pain is colic and not peritonitis. On the contrary, we may announce that there exists pleurisy or peritonitis, as the case may be, if the thermometer rise to, or above 38°, or to 38°.5 during the first hours of the attack.

I shall now resume my subject—paracentesis of the chest.

The necessity for performing paracentesis in those cases of excessive pleuritic effusion, in which there is a danger of the occurrence of sudden death, is now admitted by all real practitioners; but its utility, nay, its necessity, is also beyond doubt, when the object is to ward off those accidents, which, as I stated at the beginning of this lecture, may arise from continuance of the effusion. In such cases, it is often the only means of preventing a fatal issue, or of prolonging life.

Experience shows that even in the simplest case of pleuritic effusion, resolution requires a long time, even when the effusion is small in quantity. We all know how great a difference there is in this respect between pneumonia and pleurisy: the march of the former is as much characterised by rapidity, as is the march of the latter by slowness, at least in the decline of the attack. All practitioners have been struck with this fact; and it is not unusual to see patients—even when treated most energetically and most rationally—retain for a month, for two months, or for a longer period after the termination of the acute stage, obscurity in the respiratory sound, and pleuritic blowing, testifying not only to the existence of false membranes, but also to the presence of a certain quantity of the effusion. Suppose, Gentlemen, that you had to do with a case in which the quantity of fluid effused was not small, but, on the contrary, very considerable, you can understand why resolution should be necessarily slower in the one case than in the other. Suppose, for example, that the pleura contains two or three litres of serosity, it would not be surprising for three, four, five, six months or longer to elapse, before the effusion had entirely disappeared:

this greater slowness in the absorption is perhaps as much dependent on the pressure exerted by the excessive quantity of fluid upon the serous membrane by which absorption has to be performed, as by the mere greatness of the quantity. The effusion does not remain harmlessly in the cavity within which it is enclosed—it produces febrile action; and the longer the effusion remains, the longer will the patient continue in a feverish state. The nutritive functions will be disturbed; for, as has been clearly shown by the beautiful experiments of M. Cl. Bernard, there is a sufficient cause of fever, whenever digestion is badly performed, whenever the gastric secretions lose their physiological properties, and become unfit to accomplish those operations in vital chemistry which it is their office to perform in the process of chymification. Fever continuing from the presence of pleuritic effusion, will ultimately exhaust the individual, causing him to sink in a hectic state. This hectic fever invariably occurs where there is suppurative pleurisy, or *empyema*, as it is called. Although serous pleuritic effusion, very great in quantity, may exist for a long time without becoming purulent, cases occur, particularly in children, in whom this transformation takes place more or less, the pleurisy remaining simple, at least to the extent of not being expressive of any diathesis.

Gentlemen, as you are aware, at the beginning of an inflammatory affection of a serous membrane, the microscope scarcely discloses any of the constituents of pus; but if the malady continue, the microscope will enable us to see pus globules, which will go on becoming more and more numerous as the inflammation advances. The pleural serous membrane, when it has been for a long time the seat of inflammation, at last secretes pus, just like the cutaneous and and mucous membranes. At the beginning of a bronchial catarrh, there is no pus in the sputa; but in a short time, the expectoration becomes muco-puriform.

It is, therefore, our duty, not to allow an inflammation of the pleura to go on too long, otherwise we shall see an effusion become purulent, which originally was serous. This fact ought to cause us to decide to operate in cases in which there is a large quantity of effusion; for as I have just said, these are the cases in which resolution will be slowly accomplished.

There are not only pleurisies which become purulent from the mere continuance of the state of inflammation, but there are also pleurisies which from their nature are purulent from the first.

These suppurative pleurisies now solicit our attention for a few minutes.

In virtue of a special condition of the system, a condition which often results from the puerperal state, and is also induced by the eruptive fevers, by smallpox, but still more frequently by scarlatina, inflammatory affections of serous membranes—the serous membranes which cover the great splanchnic cavities and the synovial membranes of the joints—have a very great tendency to become suppurative.

For example, in women who have been recently delivered, an articular affection which in any other state of the system would have been nothing more than simple arthritis, at once becomes a purulent arthritis. A pleurisy, which in ordinary circumstances would have been a simple pleurisy, becomes purulent. Those of you who have read Dr. Charrier's thesis know how common these suppurative pleurisies were in 1854.[1]

A short time ago, we received into our wards a woman who had been confined eleven days previously in the Maternity Hospital. On the very day of her return to her lodgings, she was seized with pain in the side, shivering, and intense fever. On the fifth day from this attack, when she came to the Hôtel-Dieu, I found that there was pleurisy of the left side. The necessity for tapping very soon became urgent: the operation was performed on the ninth day from the beginning of the symptoms, when there was drawn off a dirty-looking fluid resembling thick broth. I have no doubt that it would have been found to contain the constituents of pus, had it been examined by the microscope. After some days, there was a return of the effusion: in about a fortnight, the place where the puncture had been made reopened of itself, and gave exit to a large quantity of fœtid pus. The woman died. At the autopsy, we found, on opening the thorax, that the pleural cavity communicated with the wound made by the trocar, and was filled with gas and fœtid pus; on the interior surface of the lung, between the two lobes, there was a circumscribed pleurisy, which formed a sort of cyst containing nearly two hundred grammes [about twenty-five fluid ounces] of pus. It is evident that the suppuration could not in this case be attributed to the puncturing of the chest, as the suppuration had taken place in the encysted pleurisy, as well as in that which occupied the great pleural cavity originally emptied by the paracentesis.

[1] CHARRIER:—Sur l'Epidémie de Fièvre Puerpérale Observée en 1854 à la Maternité de Paris.

The purulent pleurisy was produced under the influence of a special diathesis, or suppurative tendency which exists in puerperal women, and is well known to all physicians.

As I have already reminded you, the same thing takes place in eruptive diseases. You know—and I mentioned the circumstance, when lecturing on smallpox—that in that disease, the slightest inflammation is very apt to become suppurative. A very frequent sequel of confluent smallpox is the formation of numerous abscesses in various parts of the body, which go on forming for six weeks, two months, three months, or even longer. Patients who have escaped terrible attacks of the disease itself, sink from these abscesses, exhausted by interminable colliquative suppuration.

But it is principally as a sequel to scarlatina, that the suppurative tendency shows itself in serous and synovial membranes. Thus, scarlatinous arthritis, generally particularly mild, and much shorter in its duration than ordinary articular rheumatism, assumes in some cases, a very violent character, terminating in death: and when at the autopsy, the joints are opened, they are found to be filled with pus. In these cases, we also meet with suppurative pericarditis. Finally, the suppurative tendency shows itself by purulent effusions into the cavities of the pleuræ.

I directed your attention to this important point when lecturing on scarlatina. To the cases which I then brought under your notice, let me add the following.

On the 9th September, 1849, a boy six years of age, who had been in a very alarming state from the end of August, was brought to my wards at the Hôpital des Enfants Malades. On the 20th of that month, he was seized with scarlatina; the attack seems to have been very serious. I was at once struck with the general anasarcous appearance presented by the patient: I discovered extensive effusion in the left side of the chest. A large blister was immediately applied, and an infusion of digitalis was ordered to be taken in a tisane. At the end of eight days, his state was much worse. The poor child, sitting up in bed, supported by pillows, was panting for breath, and with difficulty answered in monosyllables the questions addressed to him. His face was of a livid blue; and the extremities were cold. The pulsations of the heart were quick and small: the pulsations of the radial arteries could no longer be felt; and everything seemed to indicate speedy death.

There was complete dulness throughout the whole of the left side

of the chest, which was evidently distended: but the arched appearance of the chest, and the obliteration of the intercostal spaces were masked by œdematous infiltration of the subcutaneous cellular tissue. In front, where alone auscultation was practicable, no respiratory murmur could be heard. The heart was completely displaced, the apex beating at the right edge of the sternum. The tongue, white at the edges, was rather dry and rough in the middle. The motions were loose.

Paracentesis was urgently demanded: I therefore operated at once, and withdrew a litre [more that a quart] of pus. The child was immediately relieved, and was enabled to sleep, lying on his back. During the day, he was obviously much better. Immediately after the operation, the heart moved towards its position in the left side, and we heard blowing at the summit of the lung.

During the two following days, the improvement was great. The general puffiness had diminished, particularly that of the face, where lividity had given place to a well-marked rosy tint. The pleura, nevertheless, still contained a very considerable amount of effusion, and the heart, though showing a tendency to take its normal position, was still felt beating in the median line.

During the succeeding days, the general œdema sensibly decreased; but there was little change in the chest symptoms. Although the appetite returned, the general debility went on increasing. Between my visits of the 19th and 20th September, he had, each twenty-four hours, four loose motions; and this diarrhœa continued till his death, which occurred on the twenty-fourth. At the autopsy, we found the pleural cavity filled with a purulent fluid, and both the costal and the pulmonary pleura were coated with false membrane. The lung was shrunk up, and the bronchial glands contained tubercles.

Suppurative pleurisy is essentially a serious disease, and indeed generally proves fatal, the cases in which recovery takes place through the unaided efforts of nature being quite exceptional. This statement is equally applicable to cases in which the disease is purulent from the beginning, and to simple pleurisies which become purulent. It sometimes happens that the pus which has been poured out into the chest finds an outlet for itself through a perforation of the bronchial tubes. We had an example of this in a patient who lay in bed 11 of St. Agnes's ward, whom you have seen bringing up by the mouth daily large quantities of pus. To give easy exit to the pus,

it was sufficient for the patient to lean over the bed with the head down, as you have seen many times. This man had considerable hydrothorax, but was otherwise in good health. He was, on his own request, allowed to leave the hospital.

Recall to your recollection the history of a man whose case I related when speaking of the differential diagnosis of peripneumonic and pleural vomicæ—a case I saw in consultation with Dr. Bordes.

I must add, that these fortunate cases are of an altogether exceptional class, and that most frequently hydrothorax, particularly in the adult, sooner or later terminates in death.

Even in these cases of empyema, paracentesis of the chest—and this is the point I wish to come to—even in these cases, the operation renders great service. Though it certainly does not produce the undoubted benefits which it yields in excessive effusion in simple pleurisy, it at least retards the fatal termination, and in some cases leads to recovery, when there is adopted at the same time a particular means of treatment of which I shall have to speak. Of course, I do not include cases of suppurative tubercular pleurisy, nor of pleurisy in which the purulent character is dependent upon caries of bone; but even in such cases, thoracocentesis is of some use, as I shall afterwards show. At present, we have only to consider cases of pleurisy occurring under the least unfavorable circumstances—conditions which I have just pointed out.

In an interesting work, my late lamented colleague Dr. Aran, gave an account of the successful results of this mode of treatment.[1] Similar cases are given in various monographs. You will find several in the inaugural thesis of Dr. Lacase du Thiers, and a considerable number have been collected by Dr. Boinet.[2]

The following case, which occurred in my private practice, deserves to be stated.

The widow of Dr. Pauly, an estimable Parisian colleague, when suffering from the fatigue of attendance on her husband (who had died of phthisis), was seized with pleurisy in the right side, accompanied by great effusion. Chomel having been called in, recommended active treatment; but as the disease became more serious, he sent for me to perform paracentesis, if I should think it necessary to do so.

[1] Aran:—De l'Utilité de l'Association des Injections Iodées à la Thoracocentèse—in the Treatment of Purulent Effusion consecutive to acute and chronic Pleurisy, &c.

[2] *Archives Générales de Médecine*, 1853.

There was great oppression of the breathing: the effusion filled both sides of the chest. I operated, and withdrew a great quantity of somewhat muddy serosity. In two or three days, there was as much effusion in the chest as there had been prior to the operation of paracentesis. I nevertheless waited for fifteen days, at the end of which period, it became urgently necessary to repeat the operation. This time the fluid was very turbid, opaline, and evidently contained pus. I was glad that I had given exit to it. A third time, the effusion returned; and a third time it became necessary to perform paracentesis. The operation was performed by Dr. Boinet and me: on this occasion, we followed up the withdrawal of the fluid by injecting a solution of iodine into the pleural cavity. Some months afterwards, I saw the patient: she had then regained her plump appearance, and stated that she was restored to her usual health.

But it is chiefly in children that we can count on success. At a meeting of the Hospital Medical Society, Legroux and I each presented a child successfully treated for suppurative pleurisy, by a long course of injections of iodine. Let me give you an exact account of the case of my little patient.

On the 13th of January, 1853, Edme Bélize, aged six years, was attacked with pleurisy, and was treated by Dr. Fleury. Notwithstanding the most energetic treatment, the severity of the symptoms increased, and towards the end of the month, Chomel was summoned in consultation. Drs. Fleury and Chomel detected thoracic effusion which completely filled the right pleural cavity. The patient had a great deal of fever and dyspnœa. Diuretics, contra-stimulants, and cutaneous revulsives were employed with superabundant energy; but still, day by day, the effusion seemed to increase, and by the end of the month, there was general anasarca and great orthopnœa. It was under these circumstances that I was summoned in consultation.

Dr. Fleury and I being of opinion that paracentesis of the chest offered the only chance of saving the patient, immediately performed the operation, withdrawing nearly two litres of inodorous, creamy pus. Great relief followed: however, fifteen days later, the pleural cavity was again filled with effusion; and by the middle of June, the heart and liver had become displaced. The symptoms being very serious, the operation was again had recourse to, and with the same immediate good results. The pus which was withdrawn had the smell of rotten eggs.

At the beginning of July, there was a reproduction of the effusion,

but there was tympanitic resonance on the right side, as high up as the subclavicular region : succussion produced hippocratic gurgling : there evidently existed hydrothorax. We resolved to wait, but on the 15th August, there was so great an increase in the severity of the symptoms, that we decided to puncture the chest a third time, and to leave a canula in the wound, so that the treatment by iodine injections might be pursued. Upon this occasion, we withdrew nearly two litres of horribly fœtid pus, mixed with bubbles of gas. We introduced into the wound a small canula, slightly conical, three centimeters in length, having externally a button-shaped extremity : the orifice was closed by a metallic stopper which fitted as tightly as a nail fits which is driven into a hole. The stopper was withdrawn every morning, to allow the pus to flow, after which there was injected a mixture composed of nearly thirty grammes of tincture of iodine, forty grammes of water, and from twenty to thirty centigrammes of the iodine of potassium.[1]

For six months, the quantity of pus varied from 100 to 300 grammes. In general, it was not fœtid. From time to time, there was no purulent secretion : fever then supervened, and a dreadful putrid smell came from the canula.

At the end of six months, that is to say, in February, 1854, it was observed, that when the fluid was injected into the pleural cavity, it passed into the bronchial tubes, and even into the mouth of the child. The solution of iodine was then replaced by an injection of a solution of chlorine in water : afterwards, aromatic wine was used.

In each successive month, however, there was a visible diminution in the quantity of fluid : the chest was contracted, and the vertebral column was inclined to the right. Strength and appetite returned. A nutritious diet was given : also, occasionally, cinchona wine and fish oil.

Finally, in July, 1854, nearly eleven months from the date of the introduction of the canula, eighteen months from the beginning of the malady, there was almost no discharge; and by the 1st September, it had completely ceased. Upon introducing a probe, it was found that the fistulous passage had closed. The canula was withdrawn.

When I brought this case under the notice of my colleagues, the

[1] The Tincture of Iodine of the French codex is a solution of one part (by weight) of iodine in twelve of alcohol.

child was in perfect health. Respiration was heard throughout the whole of the right side: the flattening of the chest and curvature of the vertebral column, so conspicuous six months previously, were becoming less and less day by day.

In this remarkable case, purulent effusion three times necessitated recourse to paracentesis: perforation of the lung took place: a solution of iodine was injected more than two hundred times, and there were nearly as many chlorinated and aromatic injections used: in the end, however, the cure was complete.

Let me call your attention, as I called the attention of the Medical Society of the Hospitals, to the extraordinary quantity of the purulent secretion, which may be estimated at a daily average of 200 grammes for about 200 days, which is the enormous total of 40,000 grammes [more than 40 quarts].

You can understand how essential was constant and copious nourishment to enable the child to struggle with this prodigious drain on the system. I could lay before you two precisely similar cases occurring in children: one occurred in the practice of Dr. Mousset—a case of empyema following typhoid fever; and the other, in the practice of Dr. Vigny—a case of emphysema following chronic catarrh. In both of these cases, a more speedy cure was obtained than in the case the full particulars of which I have just related. The medico-chirurgical treatment adopted was similar.

I shall only cite one other example. The subject was a young lad, to whom I was called by Dr. Bonfils. He was an American, nine years of age, who, about the end of May, 1862, was attacked with pleurisy, well marked, simple, and, in the first instance, of ordinary severity. The pleurisy was on the left side. As there were no unusual symptoms, the case was considered benignant, and was treated in accordance with that impression. After some time, however, it was observed, that the effusion increased in place of diminishing, and caused the breathing to become more and more embarrassed: the fluid nearly filled the pleural cavity, and the heart was pushed out of its place.

Dr. Bonfils was called in on the 10th June; the diagnosis did not present any difficulty. The disease made rapid progress; and on the 17th June, Dr. Bonfils looking to the manner in which matters were advancing, thinking that the chest should be tapped, asked me to consult with him on the case.

The effusion occupied two thirds of the left side of the chest, and

forced the heart to the right: there was great difficulty of breathing. To hesitate was impossible: paracentesis was an urgent necessity. The operation was at once performed by my colleague, when there issued from the canula a continuous gush of pus, to the extent of 600 grammes [nearly a pint and a quarter].

This result did not surprise me: I had foreseen it, but had reserved my opinion upon the nature of the fluid. I was justified in believing that the case was one of purulent pleurisy, from the continuance of the effusion, and also because pleurisy seemed to have supervened on a attack of measles which the child had had some time previously, but from which he had not made a complete recovery.

Immediately after the operation, the breathing became easier: the vesicular murmur was heard where an instant previously it was inaudible: there was thoracic resonance, and the heart was regaining its natural position.

During the following ten or fifteen days, the effusion remaining moderate in amount, respiration seemed to be freer: the general state of the patient was not getting worse, so that there was room to hope that the malady would of itself subside. Ere long, however, unfavorable symptoms reappeared. From 17th June to 16th August, complications of the most alarming seriousness occurred: however, as at intervals, a certain amount of amelioration in the general aspect of the symptoms was observable, we remained spectators of the struggle, waiting for the occurrence of some positive indication to interfere.

About the middle of July, the patient seemed, for twelve days, to be getting into a better state, when about the beginning of August, he grew worse, and the danger became imminent.

On auscultating the chest posteriorly and laterally, there was heard a blowing sound, tubal and amphoric, the maximum intensity of which was heard in the upper part of the vertebral hollow. On percussion, a slight degree of resonance was observed in some scattered situations, which led to the supposition that the effusion was small in quantity, and that we had to do with one of those circumscribed pleurisies in which we find false membranes infiltrated with pus, rather than a collection of purulent fluid. There was the greater reason to take this view of the case, from the circumstance, that upon that side of the chest there was deformity and flattening posteriorly, while anteriorly and laterally, there was slight arching, formed by projection of the ribs, which were abnormally separated

from one another. In this situation, the dulness was complete; and no respiratory murmur was audible. The heart was out of its place, and forced upwards.

Although the state of the patient was alarming, I hesitated to interfere surgically, as the diagnosis of the local lesions was beset with causes of great uncertainty. The question was:—might not the trocar, in the event of a new puncture being made, come upon a mass of false membrane, which would prevent the flow of the fluid? However, as the deformity, the arching (which day by day became more and more decided in front), the complete dulness, and the absence of every kind of sound, made it evident that there was circumscribed effusion—as this effusion was increasing—and as the dyspnœa was very great—on the 19th August, two months after the first tapping, Dr. Bonfils saw the necessity for resorting a second time to the operation.

He withdrew 300 grammes of thick phlegmonous pus, similar to that which was evacuated on the 17th June. The operation again gave immediate relief, although there was no modification in the signs furnished by auscultation and percussion.

Some days later, at the end of August, a small fluctuating tumour formed several centimeters above the cicatrix of the wound made by the trocar: it occupied an intercostal space, the skin over which was of a violet colour. On the 1st September, this abscess was opened by the bistoury, and in this way, a pleural fistula was established, from which, for ten weeks, there was an exudation of pus, at first very tenacious, afterwards becoming serous, but never presenting an unhealthy character, and always decreasing in quantity.

From this time, the general health became satisfactory, and the cure of the local affection showed steady progress. The effusion steadily diminished in quantity, while at the same time the chest underwent that deformity which is usual under similar circumstances.

In the month of October, the patient was able to go out; and Dr. Bonfils then met him playing in the gardens of the Tuileries. Three months later, the deformity of the chest had disappeared. A few days ago, this child was brought to me in my consulting-room; and I found that his cure was as complete as possible, and that his health was excellent.

If there be one species of purulent pleurisy which seems to baffle all the efforts of medicine it is assuredly that species to which puer-

peral women are subject. The majority of such cases terminate in death, which is indeed their almost inevitable issue. Still, even in these cases, paracentesis affords a chance of recovery, as is shown by the following case.

At the beginning of 1858, Dr. Rousset did me the honour of calling me in to consult with him in the case of a young lady, who, nine days previously, had been delivered of her first child. On the fifth day after delivery, she was seized with fever and slight pain in the left side: this was the beginning of a pleuritic attack. Dr. Rayer was called in: treatment, at once the most active and the most rational, was resorted to. The effusion advanced with the most frightful rapidity: on the fifth day of the disease, the symptoms assumed such intense severity, that any medical interference seemed useless. Dr. Rousset, and M. Bouley (my colleague at the Academy of Medicine and a relation of the patient), thought that possibly paracentesis of the chest might offer a chance of prolonging life. When we met in consultation, the pulse was quick and so small, that death seemed imminent: it was impossible not to see that the patient had purulent effusion. We were aware that the puerperal state imparted a serious character to the local disease: but life was ebbing, and the operation could not in the slightest degree diminish the patient's chances of recovery.

I performed paracentesis, and withdrew nearly 1,500 grammes of a turbid, semipurulent fluid. Immediate relief was experienced. The pulse regained its volume, and lost its frequency. The patient seemed to return to life, and testified her gratitude by a look which seemed to me of very good augury. There was still, however, very high fever, and some dyspnœa. Four days later, the fluid reaccumulated; and the orthopnœa became exceedingly urgent, although life did not seem to be placed in immediate jeopardy. I again tapped, the operation being performed in the intercostal space, immediately below that in which it had been previously practised: upon this occasion, I withdrew 1,200 grammes. I closed the wound in the same way as on the first occasion; being prepared to incise an intercostal space, and use injections of a solution of iodine, should the purulent secretion be renewed with similar rapidity, particularly if it assumed a fœtid character.

The case went on favorably: the fever abated: the appetite returned; as did likewise a hopeful, cheerful state of mind. During the next fortnight, I observed a slow reaccumulation of the pus.

There was after this, a little inflammatory action in the situation of each of the two punctures, and this was followed by slight fluctuation. Some days later, both wounds reopened, and yielded a large tumbler of perfectly inodorous pus. Daily, for a month, a large quantity of similar fluid was discharged. One of the wounds then closed; and for more than four months from that date, every two or three days, there was found in the bandage which encircled the patient's body, at least two or three spoonfuls of tenacious, inodorous pus.

At last, the wound finally closed. Some days after this occurred, there was dyspnœa, and increased uneasiness: then, one day, the the patient brought up by the mouth nearly a tumbler of pus. The pleural effusion, perforating the lung, had found exit by the bronchial tubes. I was not without anxiety; but I was soon reassured by seeing that the purulent expectoration rapidly diminished in quantity, and did not assume any degree of fœtor.

This vomica was not healed till the next winter, nearly a year after the first operation. Next year, the young patient went to Cauterets, whence she proceeded to Mentone, where she passed the winter: she is now about to go to Nice. At present, she is in quite as good health as before her marriage; but she easily takes catarrh, easily becomes feverish, and has sometimes œdema of the inferior extremities, which yields after some days of violent diarrhœa. The chest, which had become deformed, as a consequence of the disease, has regained its normal configuration. She generally has mucous râles, but there is nothing to lead to the belief that she has tubercles.

It is not necessary to repeat the details of a case which I referred to in my lectures on scarlatina: the patient was a child, whom Dr. Blanche and I successfully treated by paracentesis for suppurative pleurisy supervening in the course of the exanthematous fever. But as an additional example of the utility of tapping the chest in scarlatinous empyema, and as one among other similar cases which I might relate, I ask you to allow me to read an account of one of the cases which Dr. P. Brotherston, has published in the *Edinburgh Monthly Journal of Medical Science* for 1853.

In October 1853, says Dr. Brotherston, a boy, four and a half years of age, was attacked with serious chest symptoms after scarlatina. The disease was in the left side, where there was dulness and an absence of respiratory sound. The patient had a very painful cough, and œdema of the extremities. He slept badly. The appli-

cation of leeches and the administration of diuretics failed to give any relief. On the 2nd November, paracentesis was performed: the puncture was made, with a small trocar, between the seventh and eighth ribs, at an equal distance between the sternum and the spine. There was a flow of thick, yellow, healthy pus: the quantity could not be estimated. A large piece of sponge, cut out in the centre, and soaked in hot water, was applied to the orifice of the canula: eight hours afterwards, not only was the sponge saturated with pus, but pus had soaked through the child's clothes. Rapid improvement took place, and the wound closed. On the 15th November, there was distinct fluctuation in the situation of the puncture: a new opening was made, which afforded exit to ten ounces of healthy pus. The wound remained open for about a month, and discharged during the whole of that time. The child was restored to perfect health.

Before proceeding farther, I must call your attention to the fact, that in a large proportion of the numerous cases of recovery which I have brought under your notice, the pleurisy was on the right side.

In 1860, Aran published a work, from which it appeared, that when the effusion is on the right side, paracentesis produces only a temporarily beneficial effect, as the fluid re-accumulates; or, if at first, all has gone on well, in a short time tubercles supervene. It is very remarkable that Hippocrates noticed this fact without trying to explain why the probability of recovery is greater when the operation is performed on the left side.[1] Be the explanation what it may, I confess that my attention had not been called to the fact, till it was pointed out by Aran; and now that I bestow more consideration upon it, I am obliged to admit—without, however, being able to explain the circumstance—that effusions on the right side are most common in tuberculous subjects: but if Aran was unfortunate in his cases of paracentesis for effusion on the left side, you have seen, that by a chance which I cannot explain, I have cured a large number of patients with pleurisy on the right side—very serious pleurisies, giving rise to effusions enormous in quantity, and of a purulent character.

Hitherto, Gentlemen, we have been exclusively occupied with cases of suppurative pleurisy; but your surgical teachers have told you that empyema may be the consequence of a traumatic lesion of the

[1] Hippocrates:—De Morbis. Lib. ii, § 15.

chest, and they have likewise stated that these are the cases in which paracentesis is indicated.

A patient, who occupied bed 1, St. Agnes's ward, afforded us a remarkable example of traumatic empyema. The man to whom I refer was a carman, of robust constitution, who was admitted to our clinical wards on the 12th November, 1856: on admission, his malady was of six weeks' duration. He got violently squeezed and bruised between two carts, and had to be carried to his place of residence. A medical man who was called in ordered (on the day on which the accident occurred) leeches to be applied to the injured part, and on the following day, he took a large quantity of blood from the arm. These bleedings did not calm the acute pain which the patient experienced, and which continued for a fortnight afterwards. No amendment having followed the application of large blisters to the chest, this individual was sent to the Hôtel-Dieu.

On his admission, I found that there was very marked deformity of the chest, and that the right side was considerably arched. There was dulness on percussion, extending from the base of the chest to the crest of the scapula behind, and to the sub-clavicular fossa in front: in that situation, there was an abnormal degree of resonance. On auscultation, it was found that the vesicular murmur was absent in the lower part of the chest: in the infra-spinous fossa, there was a blowing sound and egophony: we also heard metallic tinkling in the infra-spinous fossa, and the sound of fluctuation produced by succussion, indicating the presence of air and fluid in the pleural cavity. The liver, pushed out of its normal position, extended far beyond the margins of the false ribs. The patient coughed a great deal: the sputa were bloody and rusty, mingled with frothy aërated matter. Respiration was rapid and painful. The pulse was small, and about 120 in the minute. The countenance was flushed and excited. The patient's strength did not seem to be exhausted. The physical signs showed beyond doubt that there was pleurisy, complicated to a certain extent with pneumonia; and also, that there existed a communication between the bronchi and the pleural cavity. Looking to the circumstances under which the malady originated, I concluded that the effusion was purulent.

It appeared to me that paracentesis was indicated. I operated—drawing off five and a half litres of thin inodorous pus. I employed Mathieu's double syringe, which enabled me, without removing the instrument, to inject 250 grammes (about half a pint) of a

solution of iodine. Decided relief was the immediate result of the operation. The patient said that he breathed freely. He lay on his right side, and went to sleep for some hours. During the night, he sweated profusely. Next morning, he did not feel so well as in the evening, although the fever had subsided.

On the third day after the operation, the 15th November, the pulse was 90, and the skin was warm and moist. On examining the affected part, I found complete dulness in the inferior half, and, on the contrary, above that, there was increased resonance. In that situation, there was neither respiratory murmur, blowing, nor egophony; and there was only to be heard a distant sound of vesicular expansion coming from the other lung. The metallic tinkling and the sound of fluctuation on succussion remained audible; the displacement of the liver continued. Respiration was tolerably free when the patient was seated, but it became laboured when he lay on the left side.

From the 15th to the 20th, nothing noteworthy occurred in his condition; but on the 20th, the dulness was found to have increased, and to have extended as high up as the third rib. The abnormal amount of resonance, and the stethoscopic signs which I have mentioned, still existed. Above the wound made by the large trocar with which I had operated, the cellular tissue was œdematous, the skin was red, swollen, and painful. During the day, the wound spontaneously re-opened, and discharged about three quarters of a litre of very fœtid pus.

Next day, the dulness did not extend so high up, and the exaggerated resonance was heard in the situation which had been previously dull. There was more fever than on the previous days. The patient had a cough which, from its frequency, was fatiguing; the sputa were rusty and very fœtid.

On the 23rd, there was a cessation in the discharge from the fistulous opening in the chest. At that date, I began to hear sounds of respiration behind, at the lower part of the lung, although there was still metallic tinkling. Respiration was freer. There was no fever.

On the 25th, the fistula, which had re-opened, gave exit to fœtid pus, which spurted out with considerable force during the fits of coughing. The physical phenomena observed on auscultation and percussion were dulness and amphoric blowing; but the vesicular murmur was heard over a greater extent, mingled with coarse mucous

râles. The general condition of the patient was satisfactory; and though he ate little, he ate with appetite.

From the 25th to the 30th November, there was a purulent discharge from the wound in the chest, which was alternately abundant and scanty; and with these alternations, the extent of the dulness on percussion varied.

On the 30th, the resonance was heard as high up in front as the fifth rib, and behind nearly to the angle of the scapula. The vesicular murmur was audible throughout all the upper part. The strength and appetite of the patient were good. He sat up out of bed during the day.

From the day of the operation, we had encircled the base of the chest with a girdle made of broad bands of diachylon, which were renewed daily. During the whole of December, the patient made visible progress; but I do not find anything which requires to be specially mentioned.

On the 10th January, 1857, the dulness continued as high up behind as the angle of the scapula. The vesicular murmur, still feeble and accompanied by mucous râles, was heard even down to the bottom of the lung; it was everywhere distinctly heard. The right side of the chest was remarkably constricted. For twenty-four hours, there was not the least exudation from the wound, which seemed to be quite cicatrised.

On the 23rd January, the patient, who had for some time been on full diet, and eating his entire allowance, asked permission to leave the hospital. He left; retaining no remains of his malady, except a slightly fœtid expectoration. He promised to come to show himself from time to time. He came back for this purpose on the 30th: his condition was then excellent, although there was still some dulness posteriorly, where the feebleness of the respiratory murmur indicated that all was not yet right. Fifteen days afterwards, on the 13th February, he again returned to see us, when he stated that he had resumed his occupation as a carter.

Gentlemen, I have stated that pleurisy, when the effusion has been long present, may become purulent, the pleurisy remaining simple, that is to say, not being the expression of any diathesis, and I have told you, that this is particularly observed in children: I have also remarked, that constant determination towards the

thoracic organs may lead to the development of tubercles in predisposed persons.

Whenever chronic inflammation is developed without any known cause, or in consequence of a traumatic exciting influence, in individuals under the dominion of the tuberculous diathesis, the manifestations of that diathesis show themselves in the affected organs and tissues. Suppose, for example, a lad, the child of scrofulous parents—a subject in whom there is reason to fear that scrofula exists, though it has never shown itself—suppose that this lad sprain a joint, it is necessary to be very careful, and to watch the injury much more closely than in an ordinary subject, as there is a risk of the sprain becoming a white swelling: there is a similar danger in respect of abdominal and thoracic inflammations in the scrofulous. In a child of good constitution, born of healthy parents, chronic diarrhœa would not bring with it the same dangers as in strumous or tuberculous children. In strumous and tuberculous subjects, the diarrhœa continues a long time, the intestinal inflammation is persistent, involving the glands of Peyer and the mesenteric glands. You will, under such circumstances, see the affection known by the name of tabes mesenterica [*carreau*] ; or, perhaps, the intestinal inflammation, from its contiguity to the peritoneum, will give rise to chronic inflammation of that serous membrane, and to tuberculous granulations. Likewise, in persons under the dominion of the strumous diathesis, when a pleuritic effusion is of long standing, the inflammatory determination towards the pleura will call forth manifestations of the diathesis in that serous membrane, precisely as enteritis, peritonitis, and arthritis, are called forth in the mesenteric glands, peritoneum, and joints.

From these considerations, then, it follows, that paracentesis of the chest ought to be performed, with the least possible delay, in cases of great pleuritic effusion.

Gentlemen, these extensive pleuritic effusions coming on slowly—
—these *latent pleurisies*—are frequently themselves *manifestations of the tuberculous diathesis,* the expression of incipient phthisis, as was long ago pointed out by Stoll :—"*Est* (pleuritis latens) *sæpe chronica, non rarò hæreditaria, tumque in phthisin terminanda.*"[1]

These chronic effusions are not necessarily, as might be sup-

[1] Stoll :—Aphorism, 188.

posed, and as has been said, the result of tuberculous inflammation of the pleura, when we find the pleura in such cases coated with characteristic granulations. It is quite true that these appearances are often found; but it is a question, whether the tubercnlous granulations have not been developed consecutively to the effusion. It sometimes happens that these chronic effusions, even when they remain serous and limpid, are the sole thoracic manifestation of the tuberculous diathesis; as is discovered, when patients are carried off by some other affection, and at the autopsy the pulmonary apparatus is found to be perfectly healthy. Here is a case in point:—

Auguste Thillaye, aged twelve, the son of the keeper of the museum of the Faculty of Medicine of Paris, a boy of lymphatic constitution, was taken home from school, on account of severe headache and a stitch in the left side above the false ribs. He had no fever. He was put to bed, and the stitch in the side went away: next day, he drove out in a carriage. For several days, he had no appetite; but he made no complaint of pain, and was free from fever and cough.

For three successive days, his chest was examined with the greatest possible care, when it was found that he breathed equally well on both sides.

Three days later, in the evening, the left side of the chest, from base to summit, was found to be filled with fluid. A blister was applied.

Next day, there was fever for the first time. Three days later, the eleventh day from the beginning of the malady, another blister was applied, but with as little success as the first. The effusion increased; but nevertheless, the child made no complaint of pain. On the 5th, I was called in, when I expressed a wish to have Dr. Bouillaud associated with me in consultation. There was great enlargement of the left side of the chest, and the ribs were almost immovable: there was complete dulness, and we heard bronchial blowing and bronchophony. The mediastinum was pushed upwards and to the right, two centimeters from the median line: the heart was pushed over to the right side, and was felt to beat at the right nipple: the liver, and, in a still greater degree, the spleen, were displaced, both descending very low in the abdominal cavity. However, there was very little dyspnœa, but when the patient was agitated, he had some breathlessness: the pulse was 128 and small: the skin was tolerably warm: there were no gastric symptoms. I recommended

that a third flying blister should be applied ; and that calomel should be administered in small doses with nitrate of potash. For eight days, no amendment was apparent : on the contrary, the pulse became very quick, rising to 144, without any increase in the temperature of the skin : the countenance was anxious. A fourth blister was applied. I also prescribed digitalis, which was continued for eight days.

The effusion had increased : the heart beat on the right side, beyond and above the right nipple. There was neither orthopnœa, dyspnœa, nor cough. Paracentesis was resolved upon ; and was performed with the usual precautions at 10 in the morning of Thursday the 13th November. Eleven hundred grammes of serosity, greenish, limpid, and very albuminous flowed from the puncture. Immediate relief was experienced ; but, as almost always happens, the patient had frequent fits of coughing after the operation. The lung at once expanded, and respiration was heard throughout the whole of the lower part of the chest. The heart first came under the sternum, and very soon afterwards assumed its normal position.

On the 15th November, the state of the patient was good. Subcrepitant mucous râles were heard throughout the whole of the front of the chest.

On the 24th November, there was a little fever ; and some increase in the effusion.

The child made a perfect recovery from the pleuritic attack ; but he died a few months later of tuberculous meningitis. On making the autopsy, no lesion of the pleura was found, and the lungs appeared to be healthy. There were tuberculous granulations in the brain.

This case, then, corroborates the proposition I have just stated, to the effect, that hydrothorax, even when the effused fluid is purely serous, may be the manifestation of the tuberculous diathesis. It also shows, that notwithstanding the unfavourable condition of the boy's system, a condition under which he was doomed to succumb ere long, paracentesis, *necessitated* by the imminence of the danger, was of real utility, for without it, death must have been the inevitable consequence of the pleuritic effusion.

When pleuritic effusion accompanies pulmonary manifestations of the tuberculous diathesis—and then they are generally purulent—paracentesis is useful in those cases in which the largeness of the

quantity of the effusion is in itself a formidable complication. Assuredly, the existence of tubercles, and still more the existence of pulmonary cavities, leaves but little chance of the operation proving successful; but if we cannot hope to obtain from it an absolute cure, in consequence of the fatal character of the principal and dominating malady, we can at least prevent imminent death, and considerably prolong life by performing paracentesis. This was Laennec's opinion.[1] He said that the bad state of a lung, a lung filled with tubercles, ought not absolutely to forbid operating for empyema, not even when pectoriloquy is audible in the summit of the lung compressed by the effusion, if the other lung seem to be sound. In such a case, Laennec considered that a cure was possible.

From cases observed by reliable authors, it appears that paracentesis may even be useful when there is *hydro-pneumothorax*.

Many years ago, I operated, at an interval of some weeks, upon two individuals who were both patients at the same time in our clinical wards.

One of these persons was a Piedmontese, aged twenty-six, who was by profession a juggler. Generally enjoying good health, but from his occupation necessarily leading a very irregular sort of life, he attributed the malady for which he sought our aid to a chill got in coming from an evening performance. Two months previously, the period to which he referred the beginning of his symptoms, he was tormented by a fatiguing dry cough. He continued, nevertheless, to pursue his ordinary avocations, passing from the coffee-house to the club, and from the club to the drawing-room, going late to bed, eating and drinking as usual and perhaps to excess. The only measure he adopted with a view to get rid of his catarrh was to take vapour baths and Russian baths.

Three weeks prior to his admission to the hospital—that is, three weeks prior to 3rd March 1857—he felt himself worse than usual, but still he did not keep his bed, although he felt exceedingly weak. He was losing his appetite, and after eating, was often seized with fits of coughing followed by vomiting: at night, he was exhausted by profuse perspirations: he was losing flesh: the colour of the skin was day by day becoming more and more leaden: at last, he was

[1] Laennec:—Traité de l'Auscultation Médiate. 2me édition, T. ii, p. 520.

obliged to give up his occupations, and being at the end of his resources, he resolved to seek admission to the Hôtel-Dieu.

When I saw him, he was without fever, but had a wretched appearance, characterised by great paleness, emaciation, and debility. He had hardly any cough, and only a little expectoration consisting of muco-albuminous matter without any admixture of blood. I remarked that he had the hippocratic deformity of the fingers.

On examining the chest, the physical signs of disease were found to be far from proportionate to the almost total absence of the general powers of reaction. On percussion, I found, under the left clavicle, a somewhat diminished resistance to the finger, and an increase of sound, while on the right side, there was nothing abnormal. On auscultation, supplementary respiration was heard on the right side; while on the left, respiration was of a blowing character, and accompanied by vocal resonance. Behind, the resonance was normal in the infra-spinous fossa, but it diminished from below the crest of the scapula: from this point downwards, the sound was harsh, and in the lower parts, the harshness became absolute dulness. In the infra-spinous fossa, there was double tubal blowing, most decided in expiration, during which tubal blowing alone was audible, while during inspiration, it was accompanied by, and, after fits of coughing, even replaced by, puffs of subcrepitant râles. In these situations, the voice was resonant and bronchophonic. On the right side, the respiratory sound was almost normal, except that a blowing sound was heard, which seemed to be produced at a distance from the ear, and which I regarded as transmitted from the left side.

These phenomena caused me to hesitate in my diagnosis. The absence of all the symptoms of fever, and the chronic progress of the malady excluded the idea of acute pneumonia: then, in respect of chronic pneumonia, a disease, moreover, very rare, such is not the manner in which it advances, nor are its symptoms of this character, as you can ascertain by reading Dr. Raymond's excellent thesis on this affection. On the other hand, while the general symptoms, the emaciation, the loss of strength, the impaired appetite, and the profuse nocturnal perspirations, considered in conjunction with the signs furnished by auscultation and percussion (and which might depend on cavities) suggested the idea of pulmonary phthisis, I could not harmonise the totality of the signs and symptoms with the extreme

scantiness of expectoration, and its want of special character, nor with the absence of evident signs of tubercle in the summits of the lungs. I consequently came to the conclusion, that the patient presented one of those forms of chronic pleurisy in which (as has been very well pointed out by Drs. Rilliet and Barthez) there exist extraordinary vocal resonance, cavernous respiration, tubal and amphoric blowing, and even gurgling. My colleague Dr. Béhier has recently called attention to these facts.[1]

I was thus of opinion that the patient had chronic pleurisy, and I suspected, though unable to make good my diagnosis on this point, that there was tubercular deposit on the left side, when—fourteen days after he entered our wards—the young man, whose condition had not up to this time indicated any danger, was seized in the morning with acute pain in the left side, dyspnœa, ardent fever, and a slight metallic blowing sound [*souffle métallique.*].

The acute symptoms, the pain at least, abated next day, but the dyspnœa and fever remained. There continued to be almost no expectoration. On this day, we were able to examine the patient more easily, than had been possible on the previous evening or during the previous day, from his state of anxiety and restlessness. I detected all the signs of pneumothorax: there was abnormal distension of the chest, increased resonance behind, from the angle of the scapula to the base of the lung, and an absence of thoracic vibrations on the same side. In the infra-spinous fossa, there was a metallic blowing sound, which was quite amphoric from the crest of the scapula to the base of the chest: the voice also was amphoric. On applying the ear to the posterior wall of the chest on the affected side, and percussing in front, striking a metallic pleximeter with a hammer or a piece of money, a sound was elicited similar to that produced by striking an empty barrel, or still more resembling that caused by striking a bronze vase. This phenomenon was casually pointed out by Laennec; and I have long since called your attention to it. Finally, the apex of the displaced heart was beating below the right nipple.

There was no doubt as to the existence of pneumothorax; but the signs of effusion were wanting. It was not till the 8th April—sixteen days later—that I detected them.

Day by day, the general symptoms were getting worse. From the

[1] Béhier:—op. cit., p. 611.

24th March, in addition to the continuance of fever, the excessive restlessness, and the sweating, there was dysenteric diarrhœa, which still farther increased the debility. There was still, however, little cough, and the expectoration continued unimportant in character. Five days later, I distinctly heard *hippocratic fluctuation*, when succussion was produced by an assistant, or by the patient himself.

The new diagnosis—hydro-pneumothorax—was therefore distinctly established. To my great surprise, the general symptoms improved, although the local symptoms continued. On the 29th April, the general state of the patient was apparently satisfactory. But on the 26th May, his condition had again grown worse. Although I thought of practising paracentesis, the situation of the patient did not seem to me to be so desperate as to constitute an absolute necessity for resorting to this surgical proceeding. Though far from being without anxiety as to the termination of the disease, I was afraid of accelerating a fatal issue by producing within the chest a more violent inflammation than that which already existed there; and although I felt certain of not causing death, as that expression is generally understood, I feared that I might occasion its earlier occurrence.

Nevertheless, as the patient was growing weaker, as the fever after temporarily subsiding, had become constant, I thought that it was my duty to try the operation, which (to sum up the argument in a word), however slight a chance it offered, was the only chance left. I resolved then to operate.

Believing that I had to do with a purulent effusion, I did not require to give myself any anxiety as to the prevention of air entering the pleural cavity, which moreover already contained air, as the case was one of hydro-pneumothorax. I consequently operated in accordance with the plan followed by the ancients, that is to say, by making an incision with the bistoury. Having introduced my knife between the seventh and eighth ribs, there gushed along the blade, a serous fluid which was slightly turbid, but did not appear to contain pus. I was greatly astonished, for I was expecting to see a purulent fluid: I withdrew the bistoury, that I might introduce an elastic gum sound, and in doing so gave exit to about two litres (more than two quarts) of serosity: I then injected 250 grammes of a solution containing fifty grammes of tincture of iodine and five grammes of the iodide of potassium: I allowed a certain quantity of fluid to flow, after which

I closed the wound with large bands of diachylon, of which I made a sort of girdle. The only untoward occurrence to which the operation gave rise was the formation of a serous tumour—a true subcutaneous thrombus—occasioned by the mode of operating which I had adopted: a part of the pleuritic effusion became infiltrated in the subcutaneous cellular tissue, and determined this large thrombus, which had completely disappeared in forty-eight hours, from the pressure of the bandage which encircled the body. The patient did not complain of pain within the chest, and the symptoms of absorption of iodine were very slight.

During the day, he had a decided shivering fit; but in the evening his temperature was not febrile, although his pulse was 120. From the beginning of the pneumothorax, the pulse was 120 (as in other similar cases I have met with), which ought to be attributed to the impediment to the action of the heart, occasioned by the great displacement of that organ.

His general condition improved so much, that on the 30th May, he got up, stating that he felt well. His digestion was good, and his bowels regular. Nevertheless, auscultation and percussion furnished the same signs as before the operation.

On the 4th June, there was a return of the diarrhœa, fever, and uneasy feelings. On the 7th, I found the expectoration muco-purulent and scanty. On the 22nd, the debility was increasing daily: the emaciation was great: and he had some delirium. So great had the debility become, that from this date we were not able to examine the chest, which had presented on previous days, as I have already said, the same phenomena as before the tapping; viz. distension, exaggerated resonance, amphoric blowing, metallic tinkling, hippocratic fluctuation, amphoric resonance of the voice, and the sound compared to that produced by striking a vase made of bell-metal or an empty cask.

From that date, the hectic fever never ceased; and the patient, reduced to a state of extreme emaciation, died in delirium on the 10th July at noon. On that morning, I had observed in the spittoon bloody, black, frothy, aërated sputa. In other respects, there was nothing particular in the expectoration, which was of the same nature as formerly.

On opening the dead body, it was found that the left pleural cavity was coated with thick false membrane, and entirely filled with white, creamy, inodorous pus. The lung was so closely adherent to

the vertebral column, and to the anterior wall of the chest, that it could not be removed without lacerating it; and in consequence of laceration so produced, I was unable to find the orifice of the communication which must have existed between the bronchial tubes and the pleural cavity. The pulmonary tissue was studded with tubercles in various stages: some were hard, but the majority were softening. There were numerous small cavities.

The cavity in the right pleura contained about a litre of purulent serosity. The pulmonary parenchyma was riddled with small tubercular excavations. The heart was pushed to the right, beyond the sternum; and the pericardium was lined externally with a thick layer of false membrane.

Gentlemen, though the final issue of this case was unfortunate—and the autopsy more than sufficiently explained why it was unfortunate—death could not be attributed to paracentesis of the chest having been performed, as the patient lived six weeks after the operation, which, in place of occasioning new symptoms, seemed for a time to produce amendment.

The other patient to whom I referred lay in bed 12 of the same ward. He was a man of thirty-six years of age, tall, and apparently of vigorous constitution, who had come to Paris from Berry, where he had had intermittent fever. After that illness, in July 1856, he contracted a pleurisy, which, neglected in the first instance, left behind it a great amount of effusion, which occasioned so much oppression, that he could neither speak nor drink without being obliged every moment to take breath. Two months later, on the 31st September, he saw a physician, in accordance with whose advice the chest was tapped. More than a litre of very clear water was drawn off. But the effusion was ere long reproduced: on the 25th January 1857, tapping was a second time resorted to. For two months after the second operation, the patient felt well, but the oppression then returned. The least amount of unusual exertion, such as going upstairs, or walking rather quickly, brought on shortness of breath. He experienced a constant feeling of discomfort, and painful feeling of weight on the chest, which was so much increased when he lay on the right side, that that position was impossible.

He had had cough from the beginning of his illness. For several days after each tapping, he had an increase of cough, after which, for some time, he had none at all. The cough was accompanied by a

very abundant, quite watery expectoration. At one period, when under the influence of the Eaux Bonnes, which he had been recommended to take, there was a mixture of blood-stained sputa in the expectoration, but that entirely ceased when he discontinued the use of the mineral water.

He had had a rather profuse dysenteric diarrhœa, which continued from the beginning of the illness, for four months.

The inconvenience which he experienced from the oppression of the breathing, and the daily increase of general debility, induced the patient to come to Paris for advice. He entered the Hotel-Dieu on the 9th April.

When he presented himself to me, he was apparently in a tolerably good state of body. Except a bistre tint of countenance, which recalled the aspect of those who have lived in an atmosphere contaminated by marsh miasmata, his general state seemed satisfactory.

Upon examining his chest, to which he at once called my attention, I ascertained the following facts. The left side of the chest was decidedly dilated. On percussion, the right side everywhere yielded normal resonance, except at a distance of two finger-breadths within the nipple, where there was dulness in a space of two or three centimeters from above downwards, and which, limited transversely outwards at the point I have stated, became confounded internally with the dulness of the left side. On the right side, the respiration was puerile, and exaggerated, without râles.

On the left side, in front, the body being in a horizontal position, the sound elicited was clear, from the clavicle to the nipple: but in proportion to the extent to which the trunk was raised, percussion yielded complete dulness, and when he was quite in the sitting position, the dulness extended as high up as the third intercostal space. Behind, there was complete dulness in the whole of the lower part of the chest, from the spine to the scapula downwards.

On auscultation, the vesicular murmur was very faintly heard under the clavicle and in the infra-spinous fossa, and was inaudible lower down: the cough had an amphoric resonance. During inspiration, there was also heard amphoric blowing. Finally, by an assistant percussing in front, while the ear of the observer was placed on the opposite wall of the chest, an exceedingly well-marked bell-metal sound [*bruit d'airain*] was heard. Succussion produced the sound of fluctuation [*bruit de flot*]; and this was also audible when the patient shook himself moderately. This *bruit de flot* was

heard at a certain distance, a fact of which the patient was perfectly aware.

From 9th April to 28th May, nothing worthy of being mentioned occurred. The cough was moderate, and the expectoration was mucous, devoid of any special characteristic. There was no change in the general state of the patient. He never had any fever: the pulse was small, and, it is true, beat 100 in the minute, but as its feebleness and quickness were not accompanied by heat of skin, they were no doubt dependent upon the impediment to the action of the heart occasioned by that organ being firmly pushed over to the right, where its apex was beating in the space where the dulness was observed at twice the breadth of a finger to the inside of the right nipple.

Evidently, hydro-pneumothorax existed in that situation.

Every day, the patient besought me to relieve his difficulty of breathing. I consequently resolved to perform paracentesis, and to follow it up by injections of a solution of iodine. This, in fact, was the treatment which originally suggested itself to me in this case when the patient came into my wards; but I did not then operate, as I thought that, upon the whole, the case was not very urgent, and that the operation might disturb the general state of the system, which was in an apparently satisfactory condition. However, taking into account this satisfactory state of the general system, considering the entreaties of the patient, encouraged, moreover, by the cases of cure of hydrothorax of which I have already spoken, I made up my mind: and on the 28th May I performed paracentesis.

I made an opening into the chest, by an incision of a centimeter in breadth, in the intercostal space between the seventh and eighth ribs. A purulent fluid spurted out, mingled with gas which escaped with a bubbling sound. About 150 grammes [19 ounces] of thin inodorous pus were withdrawn. Immediately after the tapping, I introduced into the wound a bent silver canula fitted with a stop-cock, and having a thin piece of caoutchouc so adjusted as to protect the integuments, and keep them from being excoriated by contact with the metallic beak. Having emptied the pleural cavity, I passed into the canula now described a gum elastic catheter, through which I injected a mixture of 50 grammes of the [French] tincture of iodine and 5 grammes of iodine of potassium, dissolved in from 100 to 120 grammes of water. I then withdrew the elastic instrument, leaving

in the chest nearly half of the injected fluid, and shutting the stop-cock of my canula, I fixed the apparatus in its proper place, by means of a diachylon bandage.

The only accident which resulted from the operation was slight subcutaneous emphysema, which disappeared in a few days. The patient, who was at first a good deal agitated by the operation, assured me, when it was over, that it had not occasioned any pain. In the evening, he complained of pain at a point corresponding to the wound made in performing the paracentesis; but he had no fever, and the pulse had even come down to 76. The heart was nearer its normal position, its pulsations being felt under the right edge of the sternum.

Next day, I withdrew from the chest a litre and a half of fluid, consisting of pus mixed with the iodine injection: it spurted out in jets with the pressure exerted upon it by the cough, which came on involuntarily.

On 30th May, a second injection, similar to the first, was thrown into the pleural cavity, after I had evacuated by the wound about a litre of purulent fluid containing some sanguinolent striæ.

The injection was repeated on the 2nd and 4th June.

The general state of the patient continued good, and from day to day the flattening of the thorax proceeded: from day to day, also, less fluid was withdrawn when the canula was opened: a certain quantity escaped during the 24 hours, running down the walls of the chest: but on the 6th June, only a few spoonfuls were collected. The fluid preserved its purulent character, and remained inodorous: on that day, the fifth, and a few days later, the sixth injection was made. These injections were repeated every three or four days, up to the 28th July, at which date the seventeenth injection was performed. The patient suffered no inconvenience from the injections except a sensation of heat in the chest. The only symptoms of iodism which he presented was a certain amount of itching, and, on one occasion, the taste of iodine in the throat.

The chest became more and more flattened; but the signs on auscultation remained nearly the same, till the 12th June. On that day, I heard, under the left clavicle, some rather coarse mucous râles which were not displaced, but rather increased, by coughing. The effusion was really diminished, but the capacity of the thorax was lessened by the great flattening of its walls which had taken place, while the heart, gradually reoccupying its normal position, was now

beating on the left side, its apex still remaining, it is true, three finger breadths to the inside of the left nipple. On the 17th June, an instrument made of gum was substituted for the silver canula. On the 25th June, the gum sound was finally removed, as the wound remained sufficiently open to allow an instrument to be introduced when an injection had to be made.

A remarkable auscultatory phenomenon was present, a phenomenon to which I have often called your attention. This was a sound heard posteriorly over the infra-spinous fossa, a blowing sound so soft, and so velvety [*tellement en nappe*] if I may venture to use such an expression, that it might be mistaken for the normal respiratory murmur: it was not, however, exactly the vesicular murmur; and in this situation, there was exaggerated thoracic resonance.

The general condition of the patient presented alternations of amendment and retrogression. On the 12th June, he was seized with diarrhœa, which continued for a fortnight to resist treatment with chalk, bismuth, and nitrate of silver, but which at last yielded to a pill taken twice a day consisting of 005 of ipecacuan, 0,005 of extract of opium, and 001 of calomel.[1] Though the diarrhœa reduced his strength, he retained his appetite. Under the use of cinchona wine and a tonic regimen, he regained strength. On the 28th July, enchanted with his condition, he was boasting of ascending the stairs of the hospital without experiencing fatigue, or being much winded. Somewhat copious perspirations, the occurrence of which was coincident with the diarrhœa, were now less considerable.

The amendment, nevertheless, did not continue; and I was obliged, at intervals, to return to the use of the iodine injections, which were in all employed forty-two times. In the beginning of the following year, he became the subject of hectic fever, by which he was carried off on the 28th February. At the autopsy, we found tubercles in the lungs.

In 1853, I submitted the history of a similar case to my colleagues of the Medical Society of the Hospitals. The patient was a woman aged 34, who when she came into my wards, had all the signs of hydro-pneumothorax: the oppression in the breathing was so great that death seemed imminent. I performed the operation by incision

[1] Five centigrames (005) is about 5 sevenths of a grain; and one centigramme (001) is about one seventh of a grain. Five milligrammes (0,005) is about 5 seventieths of a grain.

for empyema. As in the first of the two cases which I have now narrated to you, the fluid removed was limpid and transparent, and the gases were perfectly inodorous : but on the third day after the operation, the fluid had become fœtid : I then injected a solution of iodine, which occasioned neither pain nor feverish reaction. Seven days later, erysipelas supervened at the base of the chest, under the diachylon bandage which had been applied. I nevertheless repeated the injection, using, however, a weaker solution : in the evening, some symptoms of the toxic action of iodine showed themselves. The erysipelas progressed, and invaded the edges of the wound. Fifteen days after the tapping, the patient died.

At the autopsy, the pleura was found coated with a layer of pultaceous purulent matter, which was easily scraped off with the back of the scalpel. The lung was so shrunken as not to occupy more than the two upper thirds of the vertebral hollow. We found the perforation. There were tubercles in this lung; and in its centre, cavities which contained neither pus nor blood.

The paracentesis cannot be considered as having been the cause of death in these two cases, any more than in the first which I described : in both the men who were patients in Saint Agnes's ward, death occurred at a long interval after the operation, and in the woman of Saint Bernard's ward, the erysipelas of the trunk having begun in a situation remote from the trocar wound, was a complication independent of that wound. Moreover, in the last-mentioned case, paracentesis was the only possible means of preventing death, which the impediment to respiration rendered imminent.

Hence, Gentlemen, in hydro-pneumothorax, even when associated with tubercles in the lungs, the practitioner ought to intervene surgically, if the evolution of gas and the effusion of fluid threaten to produce suffocation. Some physicians who deny the utility and necessity of tapping the chest in cases of excessive effusion, simple or purulent, say that the operation proves beneficial when the hydrothorax is complicated with bronchial fistula. This, you observe, is to go far beyond what I teach : for while I hold that paracentesis is necessary in cases in which there is a large quantity of effusion without any complication—while I consider that it is useful in such circumstances, especially in children, in whom the effusion is apt to be purulent, I make reservations in respect of hydro-pneumothorax, particularly when it is symptomatic of pulmonary tubercle. When the question of performing paracentesis arises in such cases, I hesitate

very much to operate; but I grant that even in such cases, though tapping does not cure, it gives great relief and prolongs life.

Dr. Hughes, physician to Guy's Hospital, London, mentions a case in which he accomplished a cure after two tappings. The patient having succumbed long afterwards under the progress of the tuberculous disease, which showed itself in the other lung, it was observed at the autopsy that cicatrisation had taken place on the side first affected.

Cancer of the pleura may be accompanied by effusion in such quantity as to necessitate paracentesis. I need not tell you that we possess no positive sign by which to recognise during life the cancerous nature of the pleurisy in such cases.

However, if in a woman affected with cancer, or particularly in one from whom a cancerous tumour has been removed, we meet with pleural effusion slowly developing itself, we may conclude that the bronchial glands and the pleura are themselves the seat of cancerous disease: the nature of the fluid withdrawn at the time of operation by the trocar will have a great significance.

In July 1860, my friend and colleague Dr. Barth showed me a bottle containing a certain quantity of bloody fluid which he had drawn off from the chest of a patient, who had excessive pleuritic effusion. At first he was alarmed: it was not till he had reassured himself by reflecting upon the very great care with which he had examined the case, and formed his diagnosis prior to the operation, that he could divest himself of the idea of having penetrated an aneurism. I at once said to him, that he would most probably find at the autopsy a cancerous pleurisy. And it was so: there was found cancer of the lung and pleura. The only credit I deserved for this diagnosis was recollecting to have seen in my wards at the Necker Hospital, in 1844, a case of the same description—a case which I shall now relate to you.

On the 9th November 1844, a woman, aged 54, suffering from cancerous atrophy of the right breast, became my patient in the Necker Hospital. She had been several months in the Saint-Louis Hospital for rheumatic pains of the limbs, unaccompanied by any general disturbance of the system. She had some vapour baths. One day, about the 20th November, when returning from the hot-room, she felt a chill, and was attacked by an acute pleurisy on the right side, which presented nothing special in its symptoms. It was

treated by bleeding, blistering, digitalis, and calomel. About the 20th December, the effusion, so far from diminishing, was increasing. There was at that date only a very moderate degree of fever remaining. Three issues were placed on the chest. The effusion continued to increase to such an extent, that, by the end of December, it had reached the clavicle and the infra-spinous fossa of the scapula. By the beginning of January 1845, the distension of the chest had become evident: in front, the dulness soon passed the median line, and the heart was a little thrown to the left: by the 20th of that month, the dulness had passed four centimeters beyond the median line, and the heart was still more thrown out of its place: the liver was pressed down into the abdomen, and could be felt far below the false ribs. Notwithstanding this state of matters, the patient had no dyspnœa, except sometimes a little orthopnœa in the evening. There was decided fever. I perceived puffiness of the face, and infiltration of the abdominal parietes. On the 24th January, paracentesis seemed to be urgently necessary; and was then performed, in accordance with the customary rules. During the flow of the serosity, which was bloody, no coughing fits occurred. The amelioration which followed the operation was very slight: the stethoscopic signs remained unchanged. From the 1st to the 11th February, the state of the patient was nearly stationary; but at the latter date, erysipelas set in, having as its starting-point one of the issues on the chest. Notwithstanding these occurrences, the effusion having made additional progress, and threatening to suffocate the patient, I again performed paracentesis: again, I obtained a sero-sanguineous fluid. The dropsy of the chest increased, the strength failed, and soon afterwards, the woman died.

At the autopsy, I found the pleura cancerous, and covered, throughout its entire extent, with encephaloid growths.

You will find in the thesis of Dr. Lacaze du Thiers a case identical with that now described. It was observed in 1850 by Dr. Lemaitre in Professor Andral's wards: the subject was an old man. At the autopsy, cancer of the pleura and cancerous tumours in different parts of the body were discovered.

Gentlemen, changes analogous to those which take place in the pleura occur in the peritoneum. Recall to your recollection two women suffering from ascites, in whom I performed abdominal paracentesis in 1860. The effusion prevented me from recognising the presence of any tumour. From the time that the fluid began to flow

from the canula, I told you that there was cancer of the peritoneum; and the autopsy, made soon afterwards, showed me that I was not mistaken. The fluid drawn off was bloody. I also intimated that, in accordance with the law established by Dr. Barth, we should find some of the abdominal viscera affected with cancer; and at the autopsy, this diagnosis was verified.

But, Gentlemen, let me impress on you the fact, that to cause excessive pleuritic effusions to be bloody, it is not enough that cancerous productions should be disseminated in different parts of the body—to produce that result, the serous membrane itself must be affected with cancer.

In 1849, I received as a patient at the Hospital for Children, a male child eight years old, whose history is given at page 71 of the thesis of Dr. Lacaze du Thiers. This child had extensive effusion on the left side of the chest: for this affection I performed paracentesis, drawing off an amber-coloured fluid. Recovery from the pleurisy took place: but after languishing for some time, the child sunk under epileptiform convulsions, which continued for two days.

At the autopsy, small apoplectic clots were found in the brain. The kidneys, peritoneum, anterior mediastinum, pericardium, and the heart itself were invaded by cancerous products: but there was no cancerous disease of the pleura.

But, Gentlemen, the existence of a sero-sanguineous effusion does not afford absolute proof that there is cancer of the pleura or peritoneum. At the 57th page of the thesis of Dr. Lacaze du Thiers, you will find an account of a case observed by Dr. Tardieu which will prove to you that the pleura, even though not cancerous, may yield a bloody serosity. Dr. Aran communicated to me a case of the same description; and similar cases are reported by the illustrious author of the Treatise on Mediate Auscultation.

In a memorable discussion which took place in the Medical Society of the Hospitals of Paris in relation to Dr. Barth's case of which I have just been speaking, Professor Natalis Guillot referred to the history of an epidemic of measles, during which he had seen several children die of hæmorrhagic pleurisy. On the same occasion, Legroux mentioned two cases in which he had found sero-sanguineous effusion into the pleura, irrespective altogether of any cancerous diathesis.

It still remains for me to develope another reason—as appears to

me—for resorting to paracentesis of the chest in cases of extreme effusion. *In proportion to its duration, pleurisy becomes less and less curable, from the lung attracting adhesions which prevent it from regaining its place in the thoracic cavity and fulfilling its functions.*

In pleuritic effusions of long duration, the false membranes, at first albumino-fibrous, then fibro-cartilaginous, intimately soldered to each other by a cellular tissue, the product of a secondary inflammation, fix the lung to the vertebral column at the points towards which the effused fluid has pushed it. It then resists the efforts of the external air, which in the normal state contends against the natural elasticity of the organ, and tends to dilate its tissue. The lung being thus fixed, is no longer able to fill the cavity of the thorax, and that cavity is consequently narrowed and contracted by the pressure exerted on its walls by the external air.

Contraction of the chest consequent on pleurisy is a subject to which Laennec directed the special attention of physicians; and all of you are no doubt acquainted with the article which he has devoted to it in his chapter on pleurisy. He has pointed out, in a very remarkable manner, the circumstances under which this contraction takes place; and has described the anatomical state of the lung, which is so compressed and flabby as to resemble muscle, the fibres of which are so fine as to be undistinguishable. He has indicated, in a not less able manner, the signs furnished in these cases by auscultation and percussion. He then adds "that the contraction of the chest may be regarded as a real cure, inasmuch as even when it proceeds to a high degree, it not only does not render the person in whom it occurs a valetudinarian, but may even be associated with a certain amount of general vigour. Moreover, in his opinion, it does not leave any cause of a relapse, for if pleurisy is observed very seldom in cases in which the costal and pulmonary pleura are united by a great amount of cellular tissue, it ought to be regarded as impossible when the union takes place by means of a tissue so little disposed to inflammation as fibro-cartilaginous tissue."

There can be no doubt that contraction of the chest is one of the ways in which a cure is accomplished; but this mode of cure sometimes leads to incurable deformity of the chest, at least in adults: in children and young men the deformity generally decreases and ultimately quite disappears. This kind of deformity has been admirably described by Laennec.

"The subjects," he said, "have the appearance of being bent upon

the affected side, even when they try to keep themselves erect. The affected side of the chest is evidently narrower: on measuring it with a cord, a difference of more than an inch is often found between it and the sound side. Its breadth is also diminished: the ribs are in closer than normal proximity to each other: the muscles, particularly the pectoralis major, are only one half the size of those of the opposite side. The difference between the two sides is so striking, that at a first glance one would suppose that it is much greater than it is found to be on measuring. The vertebral column generally retains its straightness; but sometimes, however, it deviates a little, in consequence of the patient always leaning towards the affected side. This habit imparts to the mode of walking a peculiarity somewhat similar to limping."

This is not all: a long time elapses before the cure is complete: between the lung fixed to the vertebral column and the thoracic walls, there is a free space into which there is an interminable succession of effusions. Paracentesis prevents this; for by rapidly evacuating the serosity, it allows the lung to reassume its place almost immediately, and consequently before there has been sufficient time for adhesions to form.

The Quantity of the Effusion regulates the time at which Paracentesis is indicated.—The General Symptoms and Oppression of Breathing are Fallacious Indications.—The only Trustworthy Signs are those furnished by Auscultation and Percussion.—The Manner of Operating.—Certain Phenomena which supervene during the Flow of the Fluid.—Coughing Fits.— Flow of blood from the Wound.—The Serosity jellies in cooling, and sometimes assumes a rosy colour.—Circumscribed Pleurisies.—Objections to Paracentesis.—Paracentesis in Empyema.—Injections of Iodine; and the Permanent Canula.

Gentlemen:—I have stated the reasons which render paracentesis of the chest a necessary operation: I have told you the accidents which it may prevent, and the circumstances in which it is applicable. I ought now to set forth the indications for resorting to it.

When the accidents of which I have spoken, when the fainting fits and the lipothymia recur, when suffocative paroxysms show them-

selves, surgical intervention is urgently required; for then nothing but paracentesis can avert death. I have already supported this proposition by citing a certain number of cases. Here is another to which I beg your attention.

Dr. D., a physician, aged 35, who never had had any pulmonary affection, begun in August 1848, to experience difficulty in breathing, acceleration of pulse, and general debility. During the night, the heart beat most quickly; and dorsal decubitus was painful. Matters continued in this state for a month. My honorable colleague Professor Andral, having been consulted, detected thoracic effusion, and attributed it to a chronic pleurisy which had passed unnoticed. He recommended a large blister to be applied.

On the 13th October, Dr. D., after exposure to severe cold, was attacked by acute pleurisy on the left side. During the following days, he was three times copiously bled; and on the 25th, Dr. Andral again recommended the application of a large blister. When I was called in, the symptoms had become anxiously severe. The patient was having fainting fits; his features were distorted; and his debility was extreme. The skin was pale and cyanosed. The countenance expressed anxiety. There was considerable dyspnœa, and the respirations were 30 in the minute. The pulse was 115, and irregular. I found complete dulness of the whole of the left side. The mediastinum and heart were pushed to the right. Such being the state of the case, delay was impossible, and paracentesis was immediately performed. The operation afforded an exit to four litres of yellow, limpid serosity.

I shall afterwards return to this case, and give you some interesting additional particulars regarding it. In the mean time, I may add, that convalescence was rather protracted—that on the 2nd December, the patient began to get up, and wished at once to resume his practice, but he found himself obliged to discontinue making visits in consequence of the suffocative fits which he experienced. He went to the neighbourhood of Dieppe, where, by the use of horse exercise, he regained strength and health. From the 1st June, Dr. D. considered himself as cured: but his chest was contracted on the left side, and continued to present dulness on percussion, and obscurity in the respiratory murmur. At that date, these phenomena were perhaps more marked than they were an hour after the operation, eight months previously. Some months later, however, he felt no remaining trace of the malady, and even the deformity of the chest

had disappeared. At present, Dr. D. enjoys the best possible health.

Gentlemen, under circumstances similar to those which presented themselves in the case now described, it is impossible to hesitate; and no one will deny the absolute necessity of evacuating the effused fluid which is the cause of all the complications.

But in addition to these cases, whenever the signs furnished by auscultation and percussion reveal the presence of an extensive pleuritic effusion which may be estimated at about two litres—whenever an effusion of this description irrespective of its nature, supervenes without very marked local phenomena, without decided symptoms of reaction, it will augment rapidly—when after a certain time, nine or ten days for example, the disease has been combated more or less energetically by the ordinary therapeutic measures, and the effusion has nevertheless notably increased in quantity, the indications seem to me decisive, that the chest ought to be tapped.

When the pleural cavity is not quite full, though the operation may be free from objection and in fact offer advantages, its performance may be delayed for one or two or even for four days, always taking care, however, closely to watch the patient. In such cases, it has happened, as I have seen, that spontaneous absorption has taken place of extensive effusions, for which it had seemed that surgical interference would ultimately be necessary.

But paracentesis ought to be performed with the least possible delay, when the effusion completely fills the serous cavity, a condition indicated by absolute dulness on percussion, extending from the base of the chest in front to the clavicle, and behind, to the top of the infra-spinous fossa of the scapula, forcing out of their places diaphragm, liver, spleen, and heart. The complications which I have just been mentioning are of a threatening character.

I certainly cannot affirm that death would necessarily be the immediate consequence of this excessive effusion; but it would be impossible for me to repeat too often that there are cases of this kind, in which an unfavourable issue has been the result of delaying surgical interference and that these cases are sufficiently numerous to warrant the clinical physician to perform an operation which is not dangerous in any circumstances. Grant that the patients do not die suddenly, they will be exposed to consecutive dangers regarding which I have already spoken so fully, that it seems unnecessary to return to that subject.

One might be tempted to believe that there is no positive indication to perform paracentesis, except when the individual who had pleuritic effusion suffers from great oppression in the breathing; that there is no urgent necessity for operating, unless suffocation is imminent. Gentlemen, that is a serious mistake against which I must warn you. Oppression is one of the most deceitful of signs; and in speaking to you of the young woman who has furnished the text for the present lecture, I have called your attention to this cardinal point. It is, however, a point of so much clinical importance that I do not scruple again to insist upon it.

There are patients who, from the very beginning of the pleuritic attack, when there are hardly a few spoonfuls of fluid effused into the pleura, experience great oppression, which goes on diminishing as the effusion increases. There are others, again, in whom oppression does not supervene till the amount of effusion has become considerable, and which increases with the amount of the effusion. There are others, also, who though they have become almost suddenly affected with a great amount of hydrothorax, have never complained of the least embarrassment in respiration. It was so in the case of the woman who occupied bed 12 of St. Bernard's ward; and so it was also in the case of the man who lay in bed 19 of St. Agnes's ward.

The latter was a strong and vigorous individual, a worker in lead, who when admitted as a patient at the Hôtel-Dieu was complaining of colic. I then observed in the edges of the gums a bluish line, which seemed clearly to show that he was suffering from saturnine symptoms. He lay on his back, and did not seem to experience the slightest degree of oppression. On feeling the abdomen with the hand, I detected in the right side a moveable tumour, which descended as low down as the iliac fossa. At first, I supposed that it was composed of an accumulation of fæcal matter in the colon. The patient made no complaint of any thoracic symptoms. On examining the chest, however, I was surprised to find, on percussion, complete dulness from the base to the top of the left lung, even up to the clavicle and to the summit of the infra-spinous fossa of the scapula. The heart was squeezed to the right, and was beating beyond the sternum, even under the right nipple: the abdominal tumour was the pressed down spleen. I heard no respiratory sound.

There was, therefore, nothing in the case to lead one to suspect the presence of the enormous effusion which existed, except the physical signs furnished by auscultation and percussion. Although

the man did not seem at all inconvenienced, I thought it necessary to perform paracentesis: and I operated next morning. I withdrew more than 3,500 grammes [upwards of $3\frac{1}{2}$ quarts] of serosity, perfectly limpid and yellowish. In endeavouring to elucidate the previous history of the case, I ascertained that the effusion had begun six weeks, or two months previously. The patient recollected that he had had a chill at that period: he also remembered to have then had a slight stitch in the side, and some cough: but these symptoms did not prevent him from continuing his usual routine. In this case, recovery took place rapidly; and some days after the operation, the patient left the hospital.

Some time afterwards, my *chef de clinique*, Dr. Moynier, had occasion to tap the chest of a lad of thirteen and a half years of age, who, though he had more than two litres of serous fluid effused into the pleural cavity, did not appear to be embarrassed in his breathing.

At the beginning of April, this lad had suffered in health from hard work. The sanitary derangement consisted in a gastric affection, which yielded to rest and a purgative. Recovery, however, was not complete: the patient retained slight feelings of discomfort, and had not his usual vivacity.

On the 22nd April, he took cold from remaining inactive in a room on the ground floor; and in the evening, he had an attack of rigors, which was repeated on two successive days. He, at the same time, felt a pain in the right side of the chest, which afterwards affected also the left side going up as high as the shoulder. He had fits of coughing, unaccompanied by expectoration.

He nevertheless continued his habitual occupations, doing everything as usual, assisting his mother in the household work, and having his ordinary appetite. So little did he feel any difficulty in breathing, that on the 1st May, he carried two pails of water up to the fourth floor of the house, and six days later he walked to the Madeleine and back to his residence in the rue Lafayette.

On the 7th May—the day after this walk—he consulted Dr. Burq. He then complained of having had little inclination for food for four or five days. He was distressed by fits of coughing, which had latterly been more frequent, and by transient attacks of fever coming on in the evening, which, when over, allowed him to sleep quietly all night.

Dr. Burq having discovered that there was a large amount of effusion on the left side of the chest, brought the patient to me. I

perceived considerable arching of the walls of the chest. The ribs were raised up, and the intercostal spaces were flattened. There was an absence of thoracic vibration: behind, absolute dulness existed from the base of the chest up to the infra-spinous fossa, and in front up to the clavicle: no respiratory sound could be heard. On the right side, the vesicular murmur was exaggerated. The apex of the heart was felt to beat below and to the outside of the right mamma.

Tapping appeared to me not only to be indicated, but to be urgently required. I sent the lad to Dr. Moynier, who wished to be entrusted with the operation. Two litres of lemon-yellow serosity flowed from the canula. As generally happens—as I shall afterwards have to tell you—whilst the chest was being emptied, the patient was seized with constant fits of cough: towards the end of the evacuation, the fluid changed its character, first becoming tinged with blood, and then becoming quite bloody. This is an occurrence which I shall have to point out to you, when speaking of the phenomena which accompany paracentesis.

Proportionally as the fluid flowed from the chest, the heart resumed its place below the left mamma: sound on percussion, and also the vesicular murmur, returned to the affected side. After the operation, he had a tendency to syncope; and till the evening, he had a succession of coughing fits.

Next day, the state of the patient was satisfactory: there was still a little dulness in the lower part of the left side of the chest; and on that side, respiration was feeble. In seven days, recovery was complete.

To sum up:—When the oppression is a sign in addition to the physical signs furnished by auscultation and percussion, it has an important signification; but its absence ought not to inspire too great a feeling of security; for by refraining from interference, we run the risk of losing patients whom the operation would assuredly have saved. It is from auscultation, and still more from percussion, that we must derive our most positive indications as to the opportune moment for performing paracentesis of the chest.

Gentlemen, I now come to speak of what pertains to *the operation itself*.

In consideration of the details into which I entered when tracing the history of the question, I need now give only a very brief account of the mode of operating.

Given—an acute pleuritic effusion, for which it has been decided to operate:—How ought paracentesis to be performed?

I have already told you, that for a long time a very exaggerated idea was entertained of the danger of allowing air to enter the pleural cavity. It was at one time supposed that the entrance of a few bubbles of air into the chest would be sufficient to cause death, the notion being that the contact of the effused fluid with the atmospheric air would lead to a sort of putrid fermentation. I told you that surgeons, with a view to prevent this danger, had invented different apparatuses; and in particular I spoke to you of the instrument of Schuh. The instrument contrived by Récamier, constructed on Schuh's principle, is also upon the plan of having a valve adapted to the beak of the canula of the trocar. This valve, kept in its place by the pressure of a spring, and covered with a bit of leather, exactly resembling the key of a flute, is accurately applied to the orifice of the instrument, and can only be raised by pressure from within proceeding outwards. Ingenious though this apparatus was, much less complicated though it was than Schuh's instrument, it nevertheless presented inconveniences, not the least of which was its not being within the reach of all practitioners. The apparatus of M. Reybard, from its extreme simplicity, offered every advantage: I have explained to you this apparatus: it is that which you have seen me use, and which is employed at the present day by all operators.

Let me remark, however, that in the cases in which M. Reybard believed it to be useful, I generally do without it. It was especially for the evacuation of purulent accumulations that the surgeon of Lyons considered paracentesis of the chest necessary. In such cases, the introduction of air into the pleural cavity is an almost inevitable occurrence, and one moreover regarding which no anxiety need be entertained; because in the treatment of empyema, the canula is sometimes allowed to remain in the chest, and because in any case a fistula is formed, which establishes a communication between the pleural cavity and the external air.

When the effusion is serous, M. Reybard's canula is unquestionably useful: in such cases, indeed, it is indispensable. I am speaking, observe, of M. Reybard's canula, and not of his method of operating; for the latter is far from presenting the same simplicity as the former. Here is his operation as described by himself in his memoir published in the *Gazette Médicale* for the 16th and 25th January 1841.—The chest is penetrated, either through an intercostal space, by an incision

with a bistoury, or by boring a hole in one of the ribs by a gimlet, a very old practice which, according to M. Reybard, affords great facility for more securely fixing the canula, when it has to remain in its place for a long time. The incision in the soft parts ought to be very free, but it is specially important to make the opening in the pleura no larger than is necessary for the admission of the canula. As soon as the opening has been made, both lips of the wound in the skin have to be seized between the thumb and index finger of the left hand, while with the right hand, the operator introduces the trocar armed with a piece of sticking plaster. All these proceedings were necessary, in the opinion of the surgeon of Lyons, to prevent the entrance of air into the pleural cavity. He also gives a caution not to push in the instrument too far, lest the lung be grazed; and to protect the lung from being wounded, he says that the extremity of the canula ought to be rounded.

Such is the operative procceding recommended by M. Reybard in cases of empyema—in cases in which there is a collection of pus. If it be a fact that one can dispense with taking so much precaution, this mode of operating is not only useless but exceedingly dangerous in cases of simple pleuritic effusion, for it involves the risk of transforming the simple pleurisy into hydro-pneumothorax and empyema. In point of fact, Gentlemen, ere the canula has been twenty-four hours in the wound, it has acted as a foreign body, producing inflammation of the skin, cellular tissue, and pleura, in the neighbourhood of the opening made for it: moreover, during the efforts of inspiration and expiration, notwithstanding every care taken to prevent it, the air, pressing along the sides of the canula, enters the pleural cavity: scarcely have a few days elapsed, when it is found that there is hydro-pneumothorax, and that the serous fluid in the pleura recently so limpid, has become fœtid and purulent. It was to avoid in part this inconvenience, that M. Reybard tried to restore to favour the plan of perforating a rib: but he did not avert—he only retarded the danger. The method of operating which I have recommended, and which is now universally adopted, besides being simple, is free from danger.

The only *instruments* which are indispensable are in the hands of all medical men; viz. a bistoury, or better still a *lancet* which is much less alarming to the patient, to make the little incision, which need only involve the skin—and a common *trocar* such as is used in puncturing the abdomen, or a hydrocele. The lips of the trocar

are surrounded with gold-beater's skin, which is softened by being wetted. When gold-beater's skin cannot be obtained, a piece of the intestine of a fowl, rabbit, or cat, a bit of bladder, or a condom will serve the purpose. After tying the membranous tube to the instrument, by means of a thread, a trial is made of the working powers of this sort of valve by drawing in and blowing out alternately through the extremity of the canula opposite that which has the lips. Finally, there is required a piece of English court plaster, or diachylon plaster, cut in the form of a Maltese cross, wherewith to close the wound after the operation.

At present, there is a discussion as to the particular point where the puncture ought to be made. The question is—*What is the preferable place for performing paracentesis of the chest?*

The place which I select is (counting from above downwards) the sixth or seventh intercostal space, nearly four or five centimeters external to the outer edge of the pectoralis major.

The patient being placed in a half sitting position on the edge of his bed, the trunk supported by pillows, an assistant is entrusted with supporting the opposite side of the chest in such a way as to resist the involuntary recoil of the patient, which is apt to occur as the trocar penetrates the pleura. With the left hand, the operator renders the skin very tense, and then, with the lancet held in the right hand, he makes a puncture in the skin—only in the skin—no larger than is requisite for the admission of the trocar. This preliminary puncture is necessary; for in this respect thoracic is different from abdominal paracentesis. In the latter, there is no objection to making the perforation by one act, because the abdominal walls are wholly composed of soft parts; but in paracentesis of the chest, it is essential to facilitate the introduction of the instrument in the manner I have now described, otherwise you may incur the risk of striking the ribs with the trocar, from the patient, influenced by the painful sudden contact of the instrument, curving the chest inwards, and so diminishing the extent of the intercostal spaces by approximating the ribs. There is no risk of this occurring, if the preliminary penetration of the skin be accomplished in the manner I have pointed out. When the skin has been penetrated, you introduce the point of the trocar within the little wound, and then with a bloodless push, you easily get the instrument through the muscles and into the thoracic cavity.

Formerly, I used to recommend another manœuvre which latterly I have felt to be quite superfluous. With the view of avoiding every chance of the introduction of air into the chest, I believed that it was necessary that the external and internal openings should not be parallel. To accomplish this object, I punctured the skin below the intercostal space through which I had to penetrate, and then forcibly drew the skin upwards, so as to make my little wound in the skin correspond with the intercostal space. As soon as the operation was completed, the parts resumed their natural position, the parallelism between the two openings ceasing. But long ago I perceived that these precautions were unnecessary: the parallelism is naturally destroyed by a mechanism, which is easily understood. When the chest is distended by a great quantity of fluid, the ribs and intercostal spaces are in the same position in which they are placed by a forced inspiration, and have necessarily the relative situation which they have to the internal integument when in a state of repose: their play is under the skin, which does not follow their movements. Consequently, after the puncture, and after the evacuation of the fluid, the chest nearly or wholly regaining its normal amplitude, the ribs and intercostal spaces sink down, and as the integument is not displaced, the result is that the parallelism between the cutaneous wound and the pleural opening is destroyed. Of course it is not so completely destroyed as when my former little manœuvre is adopted; but there is no necessity that it should be destroyed: and indeed, when the effusion is purulent, want of parallelism between the two openings is an evil.

You understand, of course, that I am not now speaking of those cases in which it is necessary to leave a canula in the wound. Want of parallelism, which takes place spontaneously, would in such a case be a complication; and besides, there can be very little use in endeavouring to prevent the entrance of air during the operation, when it will generally enter of necessity by the canula at a later stage. I at present only refer to cases of acute purulent effusion treated by simple puncture. In such cases, I say that a too absolute want of parallelism might lead to troublesome consequences. Generally, after seven, eight, ten or fifteen days, a new purulent secretion has taken place, and then the pus discharges by the wound, which opens spontaneously, as you saw occur in a female patient who occupied bed 25 of St. Bernard's ward. If then, the opening in the pleura does not in any way correspond with that in the skin, the pus will burrow sinu-

ously under the integuments, causing separation of tissues and fistulæ difficult of cure.

Let us now revert to the operation itself. The trocar has penetrated the pleural cavity—a fact ascertained by feeling that its point can be moved about freely in a hollow space: the stylet is withdrawn, care being taken to open out the membrane, temporarily folded round the handle of the instrument, which has to serve as a valve: the membrane must be unfolded in such a manner as to secure the valvular action which it is meant to perform. On the withdrawal of the stylet, the fluid at first flows slowly, then in a continuous jet, and at last in jerking gushes:—I shall afterwards explain to you the cause of these differences in the modes of flowing. During expiration, the membrane is raised up by the outflowing matter; and during inspiration, it rests in exact apposition on the grooved expansion of the canula. When the flow stops, when the wished-for quantity of fluid has been obtained, the instrument is withdrawn by a quick movement: the little drops of serosity and of blood are wiped from the small wound, and the Maltese cross of court plaster, or of dyachylon plaster, is applied to it, when thus freed from moisture.

You will no doubt, Gentlemen, meet with an occurrence which has two or three times happened to me, and which you have witnessed in our wards. On withdrawing the stylet, or on your attempting to do so, not a drop of fluid passes through the canula, or if there be any flow, it is very small. This is an accident for which you ought to be prepared; for you can understand that its occurrence will occasion you disappointment and annoyance. You have in a positive manner convinced yourself that there is effusion into the pleura: mensuration and percussion of the chest have demonstrated to you that the quantity of effusion is great: you have announced to the relations that you are about to draw off three litres of water from the chest:—you introduce the trocar, and not a drop comes! How is this to be explained?

Suppose a physician performed paracentesis for the first time. His diagnosis is precise: he has accurately ascertained the position of the thoracic viscera: he has felt and heard the pulsations of the apex of the heart: he has marked the limits of the space occupied by that organ; and still, he cannot divest himself of a certain amount of misgiving. Even when the effusion is on the right side, when the heart is consequently remote from the point where the puncture has to be made, he hesitates: though he would operate boldly if he had

to perform abdominal paracentesis—a more dangerous operation than thoracic paracentesis—he stays his hand; and here is what may be the result of this hesitation.

The costal pleura is sometimes lined by layers of false membrane, which may perhaps be a centimeter in thickness. During the first eight, ten or fifteen days of the pleurisy, this pseudo-membranous layer does not adhere firmly to the costal pleura, and offers such an amount of resistance that there is difficulty in tearing it. In timidly puncturing the chest, in place of piercing right through this layer, the trocar raises it up in such a manner as to form an accidental cavity between the false membrane and the walls of the chest. If, with a view to ascertain the cause of the obstacle to the flow of the liquid, a probe is introduced through the canula, a resisting body is felt, which follows the movements of inspiration and expiration: under these circumstances, the operator cannot get rid of the idea that he has come upon the lung, and though convinced of the accuracy of his diagnosis, the frightened physician dare not continue the operation.

It is necessary in such cases, to endeavour to tear the false membrane, by using the perforator of the trocar, introduced through the canula, and pushed in more deeply, or by using a probe or a crochet needle, the latter being an excellent instrument for the purpose. Should these attempts prove unavailing, it will be necessary to make a new puncture in one of the intercostal spaces above that in which the first was made.

Specially bear in mind, that if it be necessary to proceed gently in the first stage of the introduction of the trocar, that is to say, whilst the muscles are being perforated, you must proceed quickly in the second stage, that is to say, after you have perforated them. By holding your instrument, so as to leave free about three centimeters, you have nothing to fear, for your own finger will prevent you going farther than you wish. By employing a quick manœuvre, the false membrane cannot fly before your trocar, and you will be certain to penetrate the pleural cavity.

There are other cases in which paracentesis has been performed according to rule, cases in which you have unquestionably penetrated into the pleural cavity, but in which the effusion only flows drop by drop: you have then to do with a *circumscribed*, which you must not confound with an *encysted*, pleurisy. The serous exudation is imprisoned within fibrinous partitions: these encysted pleurisies communicate with one another, or at least the fluid passes from one

into another, but it passes slowly, drop by drop. In these cases, you must endeavour to destroy, to tear, the fibrinous walls, employing the canula, a probe, or a crochet needle; and when you have done so, the flow becomes a little more free. I ought to add, Gentlemen, that cases of this description are not common.

Here, however, is a case which you will often meet with. The canula is in the midst of the effusion; but nevertheless, there is no flow. That depends upon the manner in which the patient breathes. Whether it be from a certain nervous feeling, or from a habit which he has acquired, he breathes only with the lung of the healthy side; on the other hand, the lung of the affected side, completely squeezed up against the vertebral column, contains no air, so that there is no pressure exerted from above downwards on the fluid to promote its flow. The flow does not begin till the patient is told to take deep inspirations; or better still, to strain as if at stool.

The effusion then gushes through the canula, and after a certain time dribbles out, the gush being resumed only when respiratory or straining efforts are made. The glottis being closed, the air, which cannot escape by the superior opening of the windpipe, continues to distend the lung; the capacity of the pleural cavity being at the same time diminished by the contraction of the expiratory muscles and of the diaphragm, the effused fluid, solicited from all parts to effect its exit by the opening made into the chest, escapes in jets, the spurts corresponding with the respiratory movements and the expiratory efforts.

The exertion of coughing produces similar results. Though at first it is necessary to ask the patient to cough, that is soon not required. The person who only coughed when ordered to do so, at last has frequent and involuntary coughing fits, because the lung, which has not breathed for a long time, experiences, when the air enters and opens up the air vesicles, a sort of irritation, a sort of excitement, from coming in contact with its natural stimulus, to which it had become unaccustomed.

This involuntary cough may become very violent, very frequent, and very painful, and may resist all treatment. Dr. D., whose case I related to you, complained of experiencing such severe pains when the air entered the chest as made him afraid to breathe; his respiration was short, jerking, and sobbing; and an hour and a quarter elapsed before it calmed down.

This fatiguing cough sometimes did not come on till very late in

the day. The pains which accompanied it seemed to me to depend on the tearing of the false membranes, by which the lung was adherent to the vertebral column.

Besides being useful by promoting the issue of the fluid, the exertion of coughing, the fits of cough, are beneficial by preventing syncope, when the occurrence of this complication has to be dreaded.

By chasing and driving the blood to the brain, these efforts produce a kind of cerebral plethora, which is antagonistic to the occurrence of syncope.

Towards the completion of the operation, the fluid, which flows from the canula, generally presents changes of colour. The serous effusion has then a red tinge from its admixture with blood, and very frequently the fluid is almost pure blood. This occurred in the young lad whose case Dr. Moynier published in the *Bulletin Général de Thérapeutique*. I saw the same in a little girl, in whose case I was consulted by Dr. Dumontpallier.

The patient to whom I refer was a girl eight years old, who neither complained of shortness of breath nor embarrassment of breathing, although for some time she had difficulty in running or going up a stair, and was tired by the least exercise. She said that she had no pain any where. Nevertheless she grew so thin, and her appetite became so flagging, that the mistress of the boarding-school where she was placed gave notice of her being an invalid to her parents, by whom she was taken home.

At that time the patient had very evident dyspnœa, yet she made no complaint regarding it. Inspiration was short and frequent: the pulse was small, wiry, and very quick. There was a small dry cough. On examining the chest, one was struck with the deformity of the thorax. On the left side, the lower ribs were prominent in front, and described a convex line more elevated than that formed by the corresponding ribs on the right side. The intercostal spaces were obviously flattened, an appearance which was rendered more evident by the emaciation of the child. Costal respiration seemed to be performed only on the right side. The antero-posterior diameter of the chest was greater on the left than on the right side. The apex of the heart did not beat under the left mamma, but beneath the sternum. When the hand was placed on the trunk, the child at the same time being made to speak, no thoracic vibrations were felt. There was absolute dulness on percussion from above downwards, in front, behind, and laterally, ascending as high

as the subclavicular and infra-spinous regions, and unaccompanied in front by any skodaic resonance. On auscultation, it was found that the respiratory murmur was entirely absent from the whole of that side of the chest; but above, along the vertebral column, in a space consequently corresponding to the bifurcation of the bronchi, a blowing sound and vocal resonance were heard. On the left side there was exaggerated resonance on percussion, with respiration puerile and supplementary, without râles or other abnormal sounds. There was evidently extensive pleuritic effusion on the left side.

Dr. Dumontpallier, thinking that paracentesis was indicated, asked me to see the case in consultation with him. There was no room for hesitation: I forthwith performed the operation. It occasioned little pain. The fluid evacuated was a perfectly limpid serosity, which at first was of a greenish-yellow, and towards the end of the flow of a red colour. There were some sanguinolent striæ which fell to the bottom of the vessel, and afterwards there came several spoonfuls of a serosity resembling pure vermilion blood. I withdrew the canula, and closed the wound by applying a piece of diachylon plaster. The quantity of fluid evacuated weighed 670 grammes.

The phenomena which followed the operation presented nothing worthy of special mention. A perceptible change very quickly took place in the condition of the child. Ten days after the tapping, she was sent to the country, and after a month's residence there, she had regained her good health. All that remained of her malady was flattening of the chest on the affected side, and this was showing a visible tendency to diminish.

The cause of the flow of blood may be lesion of the small vessels which enter into the structure of false membranes which are becoming organised. The false membranes are torn by coughing, and by the expansion of the lung; and to their laceration so caused, we must attribute, not only the slight hæmorrhage now under consideration, but also, as I have already remarked, the pains (sometimes pretty acute) of which patients complain—pains also, in part, the consequence of irritation of the bronchial tubes from the contact of air by which they have been long untraversed. The flow of blood may also be explained by supposing that, at the time when the lung opens out, the pleura, intimately united to the false membranes, is separated at some points from the lung or from the ribs, in such a violent way as to tear some of its vessels.

The fluid evacuated from the pleural cavity, on cooling in the

vessels in which it has been collected, forms into a jelly. In the more active pleurisies, it is very limpid, and presents a greenish yellow colour: and in such cases, it is not unusual to find it some hours after cooling with a rosy tint due to the globules of blood which it contains, and presenting an appearance which may be most appropriately compared to slightly tinted white gooseberry jelly.

During the operation, and as soon as a certain quantity of fluid has been evacuated, a change takes place in the plessimetric and stethoscopic phenomena. There is a return, from above downwards, of the resonance on percussion; and, at the same time, on applying the ear to the chest, the sound of vesicular expansion is heard, first at the summit of the lung before and behind, and then progressively throughout the whole extent of the diseased side. This pulmonary expansion is accompanied by mucous and subcrepitant râles, produced by the passage of the air into the vesicles, which contain mucus secreted by the surface of the bronchial tubes, and also by the unfolding of these vesicles. This unfolding sometimes gives rise to true crackling.

There has been discussion as to whether it is advantageous to evacuate at once the fluid effused into the pleural cavity. I do not understand why any inconvenience should result from doing so; and, for my own part, have never seen the slightest danger from the proceeding. The only undesirable occurrences which I have observed have been the pains and the hæmorrhages of which I have spoken—accidents of no special seriousness. I believe, indeed, that there is a great advantage in emptying the chest as completely as possible, as this is the best means of putting the lung into favourable conditions for expanding freely, and consequently of expediting the cure.

You perceive, Gentlemen, that the shorter the period during which the fluid remains in the pleural cavity, and the more complete is its removal, the greater will be the power of expansion, because the lung is entirely obedient to the pressure exerted upon it by the air, which, entering by the trachea, fills the bronchi and their ramifications, even to the vesicles. Besides, when we possess a means of cure so prompt, and so free from danger, why should we wait? I am well aware that the physicians, who, contrary to my view, think that a part only of the fluid ought to be evacuated, base their opinion on the belief that in paracentesis of the chest, as in paracentesis of the abdomen, either a too rapid or too abundant subtraction of the fluid may induce syncope.

This remark leads me to speak of *the objections* which have been urged *against paracentesis of the chest.*

It has been said that as syncope may supervene during or after the operation, it is an accident against which precautions ought to be taken. Without entering into a theoretical discussion on the point, I shall answer the objection by a statement of facts. Since I first performed, and have seen performed, paracentesis of the chest in cases of pleurisy, I have neither heard quoted, nor have I read, the history of any case in which this complication is mentioned. I admit that I once saw syncope supervene; but the occurrence took place under very peculiar circumstances, and not immediately after the operation. The case is so interesting, that I shall give you a full account of it.

During the autumn of 1848, I was called in by Dr. Bonnassies to M. L. living at Paris, 19 Quai Bourbon in the Isle St. Denis. M. L. had been gouty from his youth. So strongly marked in him was the gouty diathesis, that in addition to the chalky tophus which deformed all the joints, tophaceous concretions existed in the thickness of the skin of the hands and feet, to such a degree that the skin of these parts had the appearance of the internal surface of an aorta studded with ossific points. Two months previously M. L. had been attacked by pleurisy on the left side. The affected side was entirely filled with effusion, the heart and diaphragm being pushed out of place. For several nights, he had had suffocative paroxysms, leading to the dread of imminent death. As these attacks of dyspnœa supervened on the slightest movement, it was necessary for the patient to take very careful precautions when he made water or went to stool.

Paracentesis was decided on, and was performed. The operation presented this special feature, that at each cough the lung struck against the canula. I evacuated 2,200 grammes of a purely limpid lemon-coloured serosity. The lung unfolded; and immediately after the operation, there was heard, in the whole of the left side, the respiratory murmur, mingled with some mucous and subcrepitant râles. I ought, however, to state that the opening out of the lung was exceedingly painful: the pain continued till the following morning. M. L. declared that he was familiar with that sensation; that it differed in no respect from that which he experienced when the gout invaded the thoracic walls. There was high fever; but there was no return of the effusion, and the râles

were coarser. There was no symptom which foreboded a fatal termination.

M. L. was a man of very violent temper. Notwithstanding my formal orders to the contrary, he left his bed to go to stool. He got up, took some steps, then sat down on the convenience, and after some minutes spent in unavailing efforts, he returned to bed. Again he tried, but fruitlessly. He felt great oppression of the breathing. But he declared that he should make one more attempt. Neither the advice nor the entreaties of his family availed to induce him to desist. He resolutely got out of bed, sat on the night-stool, where for some time he made new and unavailing efforts; he then regained the side of his bed; and when attempting to step in, he expired.

When we consider this case in an impartial manner, we cannot impute death to the paracentesis; indeed, we may say that the fatal issue would have occurred sooner, if, before the operation, the patient had been placed in the same physical and moral conditions.

Syncope, then, is a very rare accident as a consequence of paracentesis of the chest, judging by the published accounts of cases in which this operation has been performed, and by the numerous cases which I have seen. No doubt it may occur, but when it does occur, is it to be attributed to the paracentesis? Ought it not rather to be attributed to the circumstances, to the organic conditions, which necessitated surgical interference, and which are not always immediately altered by the removal of the fluid from the chest?

To avoid the risk of this complication, which may prove fatal, the patients ought to be recommended to give both body and mind the greatest possible amount of repose after the operation. The same advice, however, is necessary when there exists a large amount of effusion, particularly if it has displaced the heart and large vessels.

When I communicated the case I have now laid before you to my colleagues of the Medical Society of the Hospitals, one of them asked whether the sudden death might not be attributed to rupture of the pulmonary vesicles, and the introduction of air into the veins. To that question, I would reply that this alleged rupture was either very late in occurring, inasmuch as death did not take place till the day after the operation; or, that if the rupture happened at the time when the fluid was evacuated, it is impossible to understand how the entrance of air into the veins should have been so long delayed.

It has been alleged that *sanguineous expectoration* sometimes follows the coughing fits with which patients are seized during the evacuation of the effusion. Cases of this description have been quoted; and they have been explained in the following manner. The rapid unfolding of the lung, by promoting a sudden afflux of blood from the pulmonary and bronchial vessels leads to congestion of the lung, of such an active character as to rupture vessels and cause hæmorrhage. I admit the possibility of such an occurrence, although I have never seen anything more than a frothy, somewhat rosy, expectoration; but I cannot accord to the accident that importance which the opponents of the operation would seem to attach to it.

I shall not stop longer at the other objection, viz.—that in performing paracentesis there may be a risk of *wounding the intercostal artery*.

By making, with the precautions which I have indicated, the puncture in an intercostal space—by, in the first instance, incising the skin so as to enable the pleural cavity to be penetrated without an effort—the manual operation becomes exceedingly simple, and much less liable to mishaps than bleeding from the arm, or opening an abscess—operations which we nevertheless entrust to the most inexperienced. Who, I ask, has seen these lesions of the intercostal artery? Your teachers of surgery have informed you that in sword wounds this vessel is seldom injured. The anatomical disposition of the parts explain the rarity of the occurrence, for the intercostal artery is placed in the groove of the bone, which circumstance, and the smallness of its calibre, protect it from being wounded. This, then, is an objection which spontaneously falls to the ground.

This cannot be said of other objections of which I am now going to speak. Though they admit of being very easily refuted, they are of sufficient importance to require to be discussed.

I begin with the statement to the effect, that *the operation has been useless, when tapping has been performed for effusion in acute pleurisy, and the effusion has been reproduced by the continuance of the pleurisy renewing the pleuritic secretion.*

The possibility of the reproduction of the fluid cannot be denied. Two things have to be considered in pleurisy with effusion. Pleurisy, properly so called, inflammation of the pleura, lasting for eight, ten, or fifteen days; and the effusion, which is at first under the influence of the inflammation, remains for a longer or shorter period after the

inflammation is at an end, just as a collection of pus in the cellular tissue remains after the inflammatory condition which gave rise to it has passed away. The collection of pus, or the serous effusion, are effects, the *results* of a pathological action which constitutes phlegmon or inflammation; but they must not be confounded with it.

I take for granted, that at the time when the excessive quantity of the effusion necessitated the operation, the pleurisy still continued. To give precision to the question, let me put a case with figures: I assume that the pleurisy has gone on for twelve days, and that its natural course will be to continue for three days longer: in such a case, we may possibly see the resulting effusion increased or reproduced during that period; but let us inquire what are the consequences of the surgical interference judged so opportune.

Suppose, for example, that there were three litres of effusion, and that by the tapping I evacuated two and a half. Suppose, that after the operation, one litre was secreted, the remaining effusion would only be a litre and a half, only the half of what it was originally, a quantity which might continue to exist without causing the risks incident to an excessive amount of effusion—taking no account of the fact, that we should have had not three but four litres of fluid in the pleural cavity. In place of allowing the state of the patient to become worse, we have granted time to the inflammation to terminate without inducing accidents; and also by withdrawing a portion of the fluid, we have made the absorption of the remainder a more easy process.

Moreover, Gentlemen—to continue the comparison which I have just instituted between pleuritic effusions and purulent collections—pus, when shut up in its circumscribed locality, becomes a source of inflammatory action, and is then a foreign body seeking to be eliminated from the living parts: fluid effused into the pleural cavity may likewise excite inflammation. To put an end to the inflammation occasioned by the presence of pus, the best thing which can be done is to open the abscess, and the quickest method of terminating an inflammation excited by pleural effusion is at once to relieve the pleura from the presence of the cause of the inflammation. To accomplish this object, paracentesis is unquestionably the most expeditious and certain measure which can be adopted.

I have no objection to admit that effusion may be reproduced to such an extent as to necessitate a repetition of the tapping. But what objection can there be to the repetition of an operation which

is so absolutely devoid of danger? It has been said that upon this principle, the patient, soon exhausted by successive tappings, must inevitably sink into marasmus. This inevitability is, in my opinion, anything but demonstrated. We very seldom require to repeat the operation several times in the same individual, when the affection is simple acute hydrothorax. Reproduction, when it does take place, is never to the extent of the original effusion; and generally, the fluid is absorbed. Generally, a single tapping suffices, and it is an exception to the rule to require to perform the operation twice in the same case. I admit, however, that the effusion may be reproduced to such an extent as to necessitate several repetitions of the paracentesis. Why should we pursue a different plan in pleuritic effusions from that which we adopt in ascitic effusions? Has the quantity of fluid which we withdraw from the pleural cavity, relatively small as compared to that which we are constantly in the habit of withdrawing unhesitatingly from the peritoneum, the special power of debilitating the patients and plunging them in marasmus? No one will venture to say so. The theoretical objections to paracentesis are completely refuted by the imposing mass of clinical facts which demonstrate, to all true practitioners, the chimerical character of the opposition which some adduce to the operation I defend.

Some physicians have maintained that the duration of pleurisy is prolonged in place of being curtailed by paracentesis, *the traumatic condition consecutive to tapping being*, according to them, *a new cause of inflammation of the pleura*. It is easy to refute this objection, not only by appealing to clinical facts, but likewise by referring to what has been observed in experiments made on animals, and to the recorded cases of wounds of the chest in the human subject.

When the chest of an animal is punctured by a pointed instrument, whatever number of punctures may be made, we find, on killing and examining the animal, nothing more than a little effused blood in the neighbourhood of the wounds, and traces of slight inflammation, extending sometimes around the wound to the distance of half a centimeter. You are aware of the small amount of seriousness which attaches to wounds of the chest made by puncturing or cutting instruments; and that whatever they may sometimes possess that is formidable depends on the complications which accompany them. If pleurisy has been mentioned as one of these complications, care has been taken to add that it remains local and benignant, so long as

there is neither effusion of blood nor entrance of air into the chest; and so long as no foreign body, such as a fragment of the sternum, or of a rib, fall into the pleural cavity—in which case a suppurative pleurisy supervenes.

Leaving these special complications out of account, wounds of the chest, even those inflicted by very large instruments, are free from danger; but still more exempt from risk must be the little wound of the trocar, made with all the precautions necessary to prevent the entrance of air into the pleural cavity. Interrogate the patients who have been the subjects of paracentesis, and they will all tell you that they experienced no pain in the situation in which the instrument penetrated the chest. If the effusion increase consecutive to the operation, this increase results from the pleurisy which existed prior to the operation not having been extinguished; and there is no ground for concluding that there is an exacerbation of the inflammation, as the quantity of fluid left in the pleural cavity, in place of being augmented, usually decreases.

In early times, when the operation was somewhat of a novelty, it was excusable for Stokes and Watson to entertain the fear that *paracentesis of the chest* might convert a serous into a purulent effusion, but now that experience has superabundantly demonstrated that the fear was groundless, to bring forward an argument of this description against the operation is either an indication of bad faith or of unpardonable ignorance. In this matter, Gentlemen, I appeal to such of you as regularly follow the practice of my clinical wards.

The most serious accident, and indeed the only one to be feared in paracentesis of the chest, is *the persistent entrance of air into the pleural cavity,* inasmuch as it may cause suppurative inflammation; but this danger has ceased to be a serious objection to the operation, because—thanks to the improvements in the method of performing it—thanks to the valve added to the canula—the entrance of air into the pleural cavity is no longer a possibility.

To complete my remarks upon the subject of paracentesis, I have a word to add on the *consecutive treatment,* which is just the treatment of ordinary pleurisy. To expedite the resolution of the effusion, to facilitate the absorption of the remains of the fluid not evacuated by the canula, I prescribe digitalis to be taken internally: I generally order an infusion of 50 centigrammes [nearly 8 grains] of the leaves in a litre [rather more than a quart] of water. I likewise order the affected side to be painted with tincture of iodine, the

resolutive influence of which I consider as at least as powerful as that of blisters.

Hitherto, Gentlemen, I have spoken to you only of paracentesis in cases of serous effusion. In *purulent effusion*, the operation must be performed in a different manner.

Although, from the symptoms which I have indicated, you may infer the existence of a purulent pleurisy, you can only in particular cases, attain sufficient certainty on this point, to dispense with the precautions which ought to be taken when the effusion is serous. You begin, therefore, by puncturing the chest with the trocar: you withdraw the canula, and perform the dressing as in a case of simple hydrothorax. It may happen sometimes, though seldom, that there is no new effusion; or, the effusion may recur and be evacuated by the bronchial tubes, which is a still more unusual occurrence, and one which, relatively, is very favourable. Almost, in every case, however, the purulent fluid reaccumulates, and the original wound made by the trocar, opening spontaneously, gives issue to the pus. A fistula is afterwards established, which will not close till the cure is complete, or till the pus finds an exit by the bronchial tubes, as I have just stated may occur. If, through some unusual circumstance, the pus flows only in small quantities, the chest may become flattened, at the same time that the lung resumes its place, and then the cure is accomplished without the occurrence of pneumothorax. Generally—I might say, in nearly all cases, a very large quantity of pus is discharged, and is replaced by air entering the chest: the result is hydro-pneumothorax, for which surgical interference will afterwards be required. In such cases, you enlarge the wound with the bistoury, so as to allow a larger canula to be introduced, which has to be left in the wound. The canula ought to be of metal, and bent in such a manner as not to injure the lung as it expands.

The rim of the canula is furnished with a caoutchouc ring, which, by coming between the instrument and the skin, prevents excoriation.

In cases of this description, so far from regretting a want of parallelism between the external and internal openings, as in cases of serous effusion, it is essential that the parallelism be as complete as possible. The introduction of air into the pleural cavity ceases to be a cause of dread, as you are going to endeavour to modify the condition of the diseased serous membrane by applying to it the tincture of iodine, or some other irritant fluid. The absence of parallelism

between the openings would make it more difficult to retain the canula in its place, and would also lead to the formation of abscesses and subcutaneous fistulæ. Nevertheless, it is necessary to prevent air from entering in great quantity, because its presence in the chest would impede the action of the lung, and produce irritation of the pleura of an injurious character. Hence, the incision made with the bistoury ought not to be more than just sufficient to permit the passage of the canula.

When you have made the incision, you allow a large portion of the effused pus to flow, without, however, completely emptying the pleural cavity, though this I consider useful in cases of serous effusion. You then inject a solution of iodine. The following is the formula for preparing the injection which I employ:—

Tincture (French) of Iodine	.	50 grammes.
Iodide of Potassium . . .		2 ,,
Distilled Water . . .		100 ,,

The injection consists of the above solution, with the addition of an equal quantity of tepid water.

You tell the patient to move about in such a way as to cause the injection to come in contact as much as possible with the surface of the pleura. You then allow a part of the fluid to escape, so as to prevent the iodine from producing toxic effects, which, though they might probably not be serious, ought not the less to be guarded against. You close the canula, and you put large bands of adhesive plaster round the chest.

You open the canula daily to allow a certain quantity of fluid to escape; and you repeat the injection, increasing or diminishing the quantity injected, and the proportions of the tincture employed, according to the degree in which the pleural cavity tends to contract, according also to the greater or less fœtor of the fluid which it contains, and its greater or less approximation to the character of laudable pus. The injection is then repeated only once in two, three or four days; taking care, however, to empty the chest at least once in the twenty-four hours.

It may be necessary to continue this treatment for a long time: in children, I have continued it for four, five, and even six months.

These are the cases, Gentlemen, in which we see very considerable deformities of the chest. The chest becomes flattened; and the individual is forcibly bent to the affected side, his shoulder approximating

to the base of the thorax, which presents a notable contraction, varying from two to seven centimeters. In front, there is very great flattening, and the clavicle projects: there is also flattening behind.

You understand the mechanism by which this deformity is produced. The lung, by means of false membranes, is squeezed back, and kept in contact with the vertebral column, near the root of the bronchi: when the effused fluid has been almost completely evacuated, a vacuum is produced in the chest at the moment the ribs ascend, particularly at the time the diaphragm descends: the pressure of the atmosphere then compresses the thoracic walls, whereas in the normal state, the equilibrium is maintained by the vacuum which has a tendency to be produced during inspiration being filled up by air rushing into the bronchial tubes.

This deformity, which increases, and sometimes assumes formidable proportions in young subjects, produces alarm in families. Dispel their fears: when once the effusion has been cured, the deformity will disappear. In children, although the thoracic deformity assumes, as I have said, a formidable appearance, it is seldom painful; but in adults whose bones are less easily bent, the pains are sometimes intolerable, a fact which you ought to bear in mind, so that you may not impute the sufferings of the patient to some serious lesion.

As the quantity of fluid in the pleural cavity diminishes, the lung will respond to the pressure of the atmospheric air, which will at each inspiration—that is, from twenty to twenty-five times a minute—enter the bronchial tubes. You can understand how great must be the effect of this pressure repeated an immense number of times in the twenty-four hours: you can understand how the lung, under the influence of this pressure, disengages itself from the adhesions by which it is confined, and expands sufficiently to resume, to a certain extent, its place in the thoracic cavity. The flattened ribs are approximated to the lung, having gone half way to meet it, if I may so express myself.

The thoracic deformity, then, is a condition favourable to the cure of effusion, inasmuch as it diminishes the containing cavity, while by the lung progressively expanding, the capacity of the cavity is likewise lessened, till at last, there only remains a sort of small pouch, which closes spontaneously.

In adults, and still more in old people, in whom the thoracic walls, being more rigid than in children, yield less easily to the pressure of

the atmosphere, the deformity is much less observed. This, probably, is one of the reasons why chronic purulent pleurisies, from which in childhood recovery generally takes place, are almost always fatal in old age.

Gentlemen, before concluding these lectures on paracentesis of the chest, let me mention an additional case, interesting in more than one aspect, and which is peculiarly fitted to demonstrate the advantages—to me incontestable—of this operation, as well as its harmlessness in the very cases in which it seems to be most contra-indicated on account of the complications which accompany hydrothorax.

You have all seen the subject of the case to which I refer: he was a man who lay in bed 25 of St. Agnes's ward, to which he was admitted as a patient on the 11th April, 1863. I cannot do better than read the history as written out in detail by Dr. Michel Peter, my *chef de clinique*.[1]

"A man, aged 36, was admitted to Dr. Trousseau's clinical wards on the 11th April, 1863, and placed in bed 25 of St. Agnes's ward.

"He stated that his illness had commenced three months previously, and that up to that date, he had had neither cough nor oppression of breathing. Subsequently, he had had both, as well as spitting of blood from time to time.

"On admission, he was diagnosed to be suffering from a serious lesion of the left side of the heart; viz. insufficiency of the aortic valve, probably combined with contraction of the orifice. The heart was greatly hypertrophied: and over the precordial region, the chest was arched. At the base, accompanying the second sound, there was loud though soft blowing; and with the first sound, there was blowing of a much less decided character. In other words, the signs of insufficiency of the aortic valve were much more marked than the signs of contraction. The pulse was bounding, as in cases of insufficiency. There was œdema of the inferior extremities, which had existed for three weeks.

"On the 14th May, after great oppression of breathing, the patient spat blood in large quantity. The blood was not red—not vermilion and frothy—as in tubercular hemoptysis: it was blackish, or mixed with bronchial mucus, as in pulmonary apoplexy. In fact, an attack of pulmonary apoplexy had supervened.

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[1] PETER (Michel):—*Gazette des Hôpitaux*, 13th June, 1863.

"Next day, 15th May, the patient complained of pain in the left side of the chest, of so violent a character as to cause him to utter piercing cries. On auscultation, however, nothing remarkable was heard."

"On the following day, 16th May, slight crepitation was heard when the ear was applied over the axillary margin of the scapula."

"On the 17th May, a superficial harsh, noisy sound had taken the place of the crepitation of the previous evening. There was marked dulness in the lower third of the chest. The pleuritic pain continued with diminished intensity."

"On the 18th May, all the indubitable signs of effusion were present. There was dulness in the two lower thirds of the left side of the chest, in which situation the vesicular murmur could not be heard. Posteriorly, at the junction of the upper and middle third of the chest, there was typical blowing and egophony. The pain was still very acute."

"On the 19th May, there was, posteriorly, dulness at the summit of the chest, and, anteriorly, skodaic resonance. The heart was inclined to the right of its natural place. The breathing was very much oppressed. Extreme anxiety existed, having as its causes, the cardiac disease, the turning of the heart on its own axis, profuse effusion, and continuance of the pain."

"On the 20th May, numerous causes of impediment to the function of hæmatesis, induced Professor Trousseau to have recourse to paracentesis; and the operation was, on that day performed by M. Peter, his *chef de clinique*. From the puncture, made in the sixth intercostal space and in the axillary line, there issued 2000 grammes [more than two quarts] of serosity, which, though rich in fibrin, was unmingled with blood. The fact that hemato-pneumothorax did not exist was established by there being no blood in the serosity evacuated; and that the hydrothorax was not simple was shown by the serosity being fibrous: in other words, there was a true pleurisy."

"The evacuation of the serosity was followed by great relief. But three days later, there was a recurrence of the pulmonary apoplexy, which led to the return of the pains, and a reproduction of the effusion."

"On the 25th May, the sixteenth day after the paracentesis, the effusion was as high up as the first rib. The apex of the heart was beating below the right nipple. As there was very great œdema of the lower extremities, friction with croton oil was ordered, with a

view to bring about the discharge of the serosity with which the cellular tissue was infiltrated.

"On the 29th May, thanks to the frictions with croton oil, the serosity flowed in very great profusion from the legs. The patient was altogether in a better state.

Although, however, the thoracic effusion was a little diminished, it was necessary to repeat the puncture of the chest on the 31st, when there was drawn off 1,700 grammes of a serous fluid absolutely similar to that obtained by the first paracentesis. This second operation was followed by fits of coughing, during which the lung was heard to unfold and resume its place, whilst at the same time the heart was observed to return towards its normal position: its pulsations, however, were still a little nearer than natural to the median line.

"From the date of the second operation, respiration was audible throughout the whole of the left side of the chest, though the sound was obscured by false membranes which lined the pleura. There was no return of the effusion."

"Ten days have now elapsed since the second operation was performed, and since that time a state of agonising suffering has ceased, the long continuance of which was incompatible with life, judging from the circumstance that the effusion had just been added to numerous other risks of death, suppressing, so to speak, one entire lung of a man in whom hæmatosis was already interfered with by heart disease."

"It will no doubt be observed that, without being dependent on the affection of the heart in respect of the hydrothorax, the pleurisy was in this case indirectly connected with the cardiac disease. The pulmonary apoplexy was the link which united the disease of the heart with the pleural effusion; not that there had been rupture of the pulmonary pleura and sanguineous effusion into the cavity of the chest (for the absence of colour showed that this had not occurred), but that some superficial clots had irritated the pleura, and had so determined serous exudation."

"Ought we in this case to dispute the utility of paracentesis of the chest, because there was recurrence of effusion after the first tapping? Before doing so, it would be necessary to forget that the evacuation of the fluid, in all probability, prevented the patient from dying in a state of asphyxia, or from sudden syncope. The duration, moreover, of the pleurisy was very short, if we compare its duration with that which it was natural to expect from so profuse an effusion in a man

doomed by the cardiac disease to serous infiltrations, and whose tissues were consequently in a condition ill suited to accomplish absorption."

"Perhaps there may be a reproduction of the fluid; but should the general state of the patient be ameliorated, a third tapping may prolong the life of the patient, who, had it not been for the relief afforded by paracentesis, could not have supported two extensive effusions, and still less have been able to support a third without sinking under its consequences."

Gentlemen, I entirely agree with the judicious remarks of Dr. Peter; and I do so with the more satisfaction that the patient, still in our wards on account of his cardiac affection, for which we can do nothing, is at present relieved from certain serious symptoms, for which we can do something. The effusion in his case would have proved mortal, for it was not dropsy of the pleura, the final phenomenon of cardiac cachexia, from which he suffered, but a pleurisy consequent upon pulmonary apoplexy. From a conviction of the utility, or I should rather say of the necessity, of our having intervened surgically on behalf of this man, I earnestly call your attention to his case, which, in my opinion, teaches more than one useful lesson.

In connection with this case, the editor of the *Gazette des Hôpitaux*, mentions that one of my pupils, Dr. A. Masson (of Yvetot), has published a memoir containing twelve cases in which he performed paracentesis of the chest. The note of the editor of the *Gazette des Hôpitaux* is to the following effect:—"In ten out of twelve cases, the operation was completely successful; and the author being favorably situated for keeping his eye on the patients, was able to assure himself that the cure, almost always rapidly obtained, was final. Never was the cure impeded by the slightest complication attributable to the paracentesis. In two cases only, the operation failed to effect a cure: or rather, in two cases, death occurred, notwithstanding the tapping. A woman tapped for tubercular pleurisy died of phthisis six months after the operation: and a man suffering from hydro-pneumothorax, with abscess of the lung, died after having several times vomited enormous quantities of pus.

"For most of the cases in which Dr. Masson operated, the heart was displaced by the effusion. His knowledge of the possibility of sudden death occurring simply in consequence of the greatness of the quantity of the effusion contributed not a little to divest his mind of

all hesitation as to the propriety of operating. The case detailed in Dr. Masson's memoir strikingly illustrates this terrible termination of some pleurisies.

"Dr. Masson also operated upon two patients in whom there was not much effusion; but who nevertheless wasted away rapidly, leading to the fear that a sudden outburst of tubercular disease was impending."

Gentlemen, I have thought it right to lay before you these cases derived from the practice of one of our honourable colleagues. Taken along with others which you will find recorded in medical works, they corroborate all that I have said to you of paracentesis of the chest. I shall have a feeling of great satisfaction, if I have convinced you of the vast services which this operation can render, and if I can diminish the fears which it still inspires in the breasts of some physicians.

[During the summer and autumn of this year [1869], I have had opportunities of seeing Professor Michel Peter perform paracentesis of the chest; and have also, both in conversations and in his clinical lectures at La Pitié, heard him give very able expositions of the subject. His practice strongly corroborates the teaching of Dr. Trousseau.

Professor Peter in his clinical lectures at La Pitié laid great stress upon the fact, that a continuance of the febrile state was ordinarily opposed to the absolute and immediate success of the operation. In such cases, from the inflammation not being extinct, reproduction of the effusion almost invariably takes place; and it may be necessary, according to the quantity of the effused fluid, to tap again or apply blisters. It sometimes happens that the new effusion becomes purulent; and that the original puncture in the thoracic walls becomes a fistulous opening. The patient may live, retaining this fistula for an indefinite period; or he may be speedily carried off by hectic fever. This latter termination, Dr. Peter has only seen three times in the very large number of cases in which he has performed thoracic paracentesis. In two of the three cases, the patients were highly lymphatic, though not tuberculous subjects: in the other case, the patient was rheumatic. These are facts which ought to be known.

Besides the flow of bloody serosity of which Dr. Trousseau speaks, and which is characteristic of cancerous pleurisies, Dr. Peter, in his clinical lectures, remarked, that the tapping itself might occasion the issue of bloody, or sanguinolent serosity. Dr. Peter has twice met with this form of hæmorrhage: both patients made complete recoveries. He attributes the bleeding in these cases to the trocar having torn some vessels belonging to very vascular false membranes. Blood is consequently discharged into the pleura, which renders bloody the fluid issuing from the canula. It is obvious, therefore, that there is no great cause for alarm when the fluid drawn off is bloody, provided the patient is otherwise in a good state. TRANSLATOR.]

LECTURE XXXIII.

TRAUMATIC EFFUSION OF BLOOD INTO THE PLEURA:—PARACENTESIS OF THE CHEST.

Effusion of Blood into the Cavity of the Pleura mechanically arrests Traumatic Hæmorrhage.—In such cases, Paracentesis is not only useless, but may even prove injurious.—The Blood coagulates immediately.—It scarcely irritates the Pleura.—Reabsorption takes place very rapidly.

Gentlemen:—In one of my previous lectures, I spoke to you of sero-sanguineous effusion into the pleura, recurring sometimes in an acute manner, particularly during eruptive fevers, and in a chronic form, when there is cancerous disease of the pleura. I now propose to speak to you of sanguineous collections formed in the pleura consequent upon wounds of the chest.

Although effusion of blood into the pleural cavity is a subject which belongs more particularly to surgery, and may seem to be somewhat foreign to a chair of Clinical Medicine, I think it right to go into the subject, rather than leave you in ignorance of what I know about it. I think so, because it is a pathological question in relation to which I have made many experiments, the results of which have not received adequate publicity[1]—and also, because sanguineous effusions into the pleura very frequently occasion attacks of pleurisy and empyema, thus bringing them, to a certain extent, within the domain of medicine.

What ought the physician to do, when a wound of the chest is followed by an effusion of blood into the pleura?

Many surgeons inculcate the withdrawal of the blood by suction of the wound: some have recommended tapping: and others have counselled the removal of the effused blood through an incision in an intercostal space.

[1] The results of the experiments made in 1829 by M. Leblanc and me were published in 1834, in the *Journal de Médecine Vétérinaire*.

Allow me, Gentlemen, to discuss these different proceedings; but in the first instance, let us endeavour to understand the indications which present themselves. Let us suppose that there is a great sanguineous effusion; for as yet no one has recommended active interference in cases of very limited hæmorrhage. There are two sources whence extensive hæmorrhage may arise: from an artery of the thoracic walls, or from one of the vessels of the lung. If the hæmorrhage come from one of the vessels of the walls of the chest, I cannot conceive the least benefit to result from any of the different proceedings of which I have just been speaking: I could better understand how pressure exerted on the opening by the accumulated blood might assist in the formation of a clot, and so plug the vessel. But if the hæmorrhage come from the lung, it is easy to see that the effusion itself will be one of the most important curative agencies. In proportion to the degree in which the blood is effused into the pleura, the lung is flattened and squeezed up; and at last, the cut vessels cease to bleed, because they are strongly compressed. In this way, the effusion materially assists in accomplishing the cure.

When a horse is wounded in the lung, a curious occurrence takes place. If a vessel of large calibre is cut, profuse hæmorrhage takes place into the pleura, while, simultaneously, the blood flows into the bronchial tubes; and in a short time, the animal dies. But if only some of the vessels of secondary size have been wounded, a rather profuse hæmorrhage takes place into the pleura, and on the bronchial surface: soon, however, from the accumulation of effused blood compressing the lung, the hæmorrhage ceases.

If the animal be killed soon afterwards, there is found in the lung itself, besides the effusion of which I have just been speaking, an exceedingly curious lesion, which as yet has been very ill described. In the whole course of the penetrating wound, the pulmonary cells are infiltrated with blood, and this infiltration extends from one to several centimeters. The blood effused into the cells is much blacker, and much more minutely infiltrated in the immediate vicinity of the passage made by the instrument inflicting the wound; and in that situation, there are structural changes identical with those which characterise recent nuclei of pulmonary apoplexy.

The passage made by the instrument is itself closed by fibrin, a true coagulum occupying the course of the wound, just as a blade fits into its sheaf. This protective clot is sometimes found half an hour after the wound has been made. It is imbedded in the inter-

lobular cellular tissue, or in the cells, by innumerable fibrinous roots, which break when an attempt is made to tear it out.

If the autopsy of the animal be not made for forty-eight, or seventy-two, hours after the infliction of the wound, the wound is found to be closed by a most remarkable process. The lips of the wound of the lung are inflamed, and the pleura surrounding it, to the extent of several centimeters, participates in the inflammation: a plastic exudation is then thrown out, which forms adhesions with the serous membrane, and becomes intimately amalgamated with the fibrinous mass occupying the course of the wound, to which it has become closely adherent. The wound is in this way obliterated throughout its entire course by a fibrinous clot, and its lips are covered by a fibrinous disk, adherent to the pleura, to the lips of the wound, and to the plugging fibrinous clot. It bears a considerable resemblance to a large fibrinous nail, the stem occupying the course taken by the wounding instrument, and the head being flattened upon the lung to which it closely adheres.

Gentlemen, who can fail to see that the surgeon by emptying the pleura of the blood effused into it from the wounded vessels must prevent flattening of the lung, so powerful a preventive of hæmorrhage, and must likewise frustrate the formation of that plugging clot which I have been so carefully describing to you?

Weigh well the fact, that by making an opening in the thoracic walls, you excite violent efforts at coughing, which will be peculiarly apt to increase the hæmorrhage, and to break down the plugging clot as fast as it forms.

I have been reasoning upon the supposition that an attempt is made, by the operation for empyema, to clear the pleura of the clots by which it is filled. Let us now inquire, whether it be possible to accomplish this object. I shall sum up in a few words the series of experiments by which M. Leblanc and I attempted to elucidate the question.

We made a small incision in the skin of a horse between the middle ribs: we carefully divided the tissues of the intercostal space, and when we reached the pleura, we opened it in such a way as to avoid the lung, and to limit the incision to some millimeters. By a stroke of the fleam, we then opened the jugular vein of the animal; and then, by means of a kind of funnel, the small end of which was placed in the pleura, and the other used to receive the product of the venæsection, we introduced into the pleural cavity, one hundred, two

hundred, four hundred, or even as many as three thousand grammes of blood. Having introduced the blood, we closed the wound by means of a twisted suture. In place of transmitting the blood directly from the jugular vein into the pleura, we generally received it in a syringe, and before it had time to coagulate, we injected it into the pleural cavity. We also divided an intercostal artery, and allowed a certain quantity of blood to flow from it into the pleura.

This experiment was performed on several horses. They were killed; some immediately after the operation, and others after an interval of one, two, twenty-four, forty-eight, and seventy-two, hours, and of from six to ten days. Without a single exception, however short was the interval between the injection and the autopsy, *we found the blood coagulated.* So rapidly did coagulation take place, that when, in our experiments, we opened an intercostal artery and caused the blood to flow directly into the pleural cavity, making at the same time an opening in a more dependent part of the chest, hardly a drop of blood flowed out by it. The same thing took place, when we injected from one to three kilogrammes of venous blood taken from the jugular vein, and, at the time of injection, in a perfectly liquid state.

We repeated the following experiment several times. As soon as the injection was completed, we felled the animal by a blow on the head with a hammer: we then, without a moment's delay, opened the abdomen, exposing the diaphragm: while the heart was still beating, and consequently while physiological life was still quite preserved, I opened the pleural cavity through the diaphragm, and found the blood in a clot. It was firmly coagulated, although the blood of the same horse taken at the same bleeding, but before the blood injected into the pleura and left exposed to the air in an evaporating vessel, was only partially coagulated. Let me add, that in cases in which the autopsy was made with the greatest possible celerity, not more than five minutes elapsed between injecting the blood into the pleura and ascertaining its condition there.

Gentlemen, when we received at the same time, in two separate evaporating vessels, the blood from the vein of a healthy man, and the blood from the vein of a healthy horse, it was observed that the human blood coagulated much the most quickly.

Now for the inferences from these facts. These conclusions you have already deduced. When blood is effused into the pleura, con-

sequent upon a wound of the chest, coagulation takes place in a few minutes, so that to perform the operation for empyema with a view to remove the blood is as senseless as it is useless. Whether it be suction, the worst and most absurd of all the operations, or pumping out the blood—(a still more dangerous proceeding, as it is a more forcible kind of suction)—whether simple tapping be resorted to, or whether an incision be made in an intercostal space, it will be impossible to withdraw the blood, on account of its coagulated condition.

You will, Gentlemen, nevertheless, hear it said by the most experienced surgeons, you will read in the works of the most accredited authors, that they have been able after wounds of the chest, to withdraw a great part of the sanguineous fluid by tapping, or by incision. The experiments of which I have given you an account were performed, as I have already told you, thirty years ago, by M. Leblanc and me. As you can well believe, they have been discussed and their results disputed. In the first place, it has been said that blood in contact with living parts, consequently at the same temperature it possessed when leaving the vein, does not coagulate, or at least coagulates more slowly than blood which has remained in a vessel exposed to the air, and the rapid coagulation which we described was absolutely denied, or at least otherwise explained. The experiments which M. Leblanc and I made upon the influence of temperature upon coagulation of blood taken from the vessels, experiments which have been repeated, and are at present no longer disputed, show that coagulation takes place most quickly when the blood is placed in a higher temperature. Thus, to give only a summary of our experiments:—when we received the blood of a horse in ten evaporating vessels, and placed these vessels in fluids varying in temperature from zero to 40 degrees, we ascertained that by maintaining the blood at zero, it remained fluid for several successive days, although it coagulated in less than two minutes when the evaporating saucer was kept in water at 40 degrees, and that the coagulation became slower and slower, in proportion as the temperature was lower.

Matters do not proceed differently within the pleural cavity. The blood coagulates there in a very brief space of time, because it there finds a high temperature, and the slight amount of motion communicated to it by respiration only retards coagulation by a few minutes, if it retards it at all.

Surgeons, then, have not properly understood what takes place. There is a confusion about the subject which I wish to dissipate.

The clot which forms within the pleura does not differ much from that which forms in vases where the blood is by itself. Between the two, however, there is a slight difference. In a vase, coagulation takes place more slowly; consequently, the red globules being heavier have time to be precipitated before the fibrin has contracted: the result is, that that which is called the buffy coat [*couenne inflammatoire*] composed of fibrin and serum, is always more abundant, other things being equal, when the blood remains longest in a fluid state. The clot, on the contrary, coagulates in the mass, and without forming any buffy coat, when it coagulates very quickly: that is what take place in the pleura. But after a very short time, the serosity imprisoned within the clot, partly bursts forth, and, shaken up as it were by the movements of respiration, is always mixed with a great quantity of blood globules: at the first glance, it has the appearance of fluid blood. There are two things then to be considered in cases of effusion of blood into the chest: there is the clot which generally occupies the most dependent parts—and there is the bloody serosity which comports itself exactly like the serosity of a pleurisy. If the surgeon were to tap, he would be able to withdraw a large quantity of serosity deeply coloured by the cruor; and might thus come to the conclusion that he had withdrawn fluid blood.

The quantity of this sanguineous fluid may be still farther augmented by a circumstance which I ought to mention. The presence of some blood is not a cause of much irritation, as I shall forthwith be able to prove to you, but the lesion which has caused the sanguineous effusion is a source of considerably more mischief, and very usually leads to inflammation of the pleura and lung. Matters become much more serious when there is pneumothorax. In such a case, the serous effusion comes from two sources; viz. from the clot itself, which is the least abundant source, and from the inflamed pleura, whence it is impossible to calculate how much may be exuded. In any case, the fluid secreted by the irritated pleura continually in contact with the crassamentum will dissolve a large amount of blood globules, so that if paracentesis were to be performed, it might be supposed that fluid blood was being withdrawn, whereas there is nothing evacuated but sanguineous serosity.

We have seen, Gentlemen, that making an opening into the chest, whether by tapping or by incision in the intercostal spaces, is a use-

less measure in the treatment of traumatic extravasation of blood: it would be easy for me to prove that it is, at the very least, injurious, and is frequently fatal. I have no difficulty in admitting that tapping, performed with the instruments and the precautions which are universally adopted in the present day, that is to say with a trocar fitted with the proper membranous valve, is, for the most part, a harmless operation; but there are exceptional cases, in which it gives rise to a circumscribed pleurisy, which cannot fail to be troublesome. Were there nothing in the pleural cavity but the serosity which had separated from the crassamentum, it would hardly be worth while to tap, for that serosity would soon be absorbed. If the extravasated blood—in particular, if its traumatic cause—has given rise to pleurisy, with consecutive effusion, tapping may be useful; but under no other circumstances can it prove beneficial.

As to making an incision in an intercostal space, a proceeding I adopt in cases of purulent effusion, when the effusion has been reproduced after simple tapping, it cannot be otherwise than a very dangerous proceeding in traumatic extravasation of blood.

I have said sufficiently often, that it must be useless, because the blood having coagulated, there is no possibility of the coagulum finding an exit by the opening, even were it infinitely larger than it is generally made.

Not only is the incision useless, but is far from being exempt from danger. However small it may be, it necessarily leads to the introduction of air into the pleural cavity, and this occurrence when repeated, is certain to lead to pleurisy, and hydro-pneumothorax, affections of an exceedingly serious character.[1] The blood poured into the pleura, putrefies; and it is easy to understand the risks consequent upon this occurrence. I have the most profound conviction that the majority of failures which surgeons formerly had in treating wounds of the chest, were due to this perilous proceeding, now, thank God, abandoned by the majority of practitioners. Our experiments have superabundantly demonstrated its peril.

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[1] By experiments on animals, it is easy to satisfy oneself that the accidental admission of air into the pleural cavity is perfectly harmless: but a repetition of the admission of air, even when the operation is set about with care, causes pleurisy. When a permanent opening is made in the chest, pleurisy and hydrothorax are inevitable.—See the account of our experiments in the *Journal de Médecine Vétérinaire*, already cited.

Those who still wish to try to evacuate blood extravasated into the pleural cavity, following in this respect the example of the illustrious Dupuytren, after the assassination of the Duke de Berry, are under the influence of three false ideas. They think that the blood remains fluid: they think that it irritates: and they think that it is absorbed with great difficulty. The experiments made by M. Leblanc and me demonstrate the falsity of these three suppositions.

We have already seen that the blood coagulates at the moment it is extravasated; and that we can withdraw, by tapping or incision, only the serum, without the crassamentum, a result which is really not worth the trouble.

Let us now inquire whether the blood produces irritation. In our numerous experiments, when we killed a horse, four, six, or eight days after the injection of blood into the pleura, if, as sometimes happened we found a clot, we never detected any traces of pleurisy. I grant, however, that extravasation of blood is not a perfectly harmless occurrence, and that it must somewhat irritate the serous membrane. More particularly I grant that it may predispose to pleurisy an individual who but for it would have escaped.

Some months ago, a young man was practising fencing with a friend: in an animated encounter, the knob of his adversary's foil was broken off without the occurrence being perceived; and a strong thrust penetrated the chest at the right arm-pit. There was neither external bleeding, nor subcutaneous ecchymosis, and, consequently, none of the vessels of the axillary region were injured. But scarcely had a few moments elapsed, when the wounded young man felt very acute pain in the region of the liver, exactly similar in character to that produced in the pelvis by that hæmorrhage from the fimbriated extremity of the Fallopian tube which constitutes retro-uterine and peri-uterine hæmatocele.

All the symptoms quieted down during a few days of repose. The patient had no fever; and was able without fatigue to attend a horse-race a fortnight after he received the wound in the chest. But some days later, he experienced a feeling of unease, and had some cough. Under these circumstances, I was summoned by my honorable friend Dr. Reis to meet him in consultation: we detected an extravasation of blood—not much in quantity—in the right pleura. This extravasation made rapid progress. It soon became so extensive as to suggest to me the propriety of considering whether we ought not to resort to paracentesis. A third physician was associ-

ated with us in consultation; and the result of our meeting was an adjournment of the operation. A fortnight later—ten weeks after the accident—the pleural effusion found a passage outwards by the bronchial tubes. In this way, the young man got rid of an enormous quantity of pus slightly tinged with blood, after which the expectoration diminished very gradually, and finally ceased four months subsequent to the accident.

If the traumatic hæmorrhage into the pleura may occasion a fluxionary determination, predisposing to pleurisy, will not the operation for empyema exert much more powerfully an evil influence on the state of the patient?

We have seen, Gentlemen, that the blood coagulates as soon as it is poured out in the pleural cavity; that it there excites only a very moderate amount of irritation. It is now necessary to show you that it is absorbed with a rapidity so extraordinary as to be incredible, were not the fact demonstrated by experience in the most positive manner.

When we injected into the chest of a horse 200 grammes of blood drawn from the vein; or when an intercostal artery was opened, and the blood allowed to flow into the pleural cavity, there were in the majority of cases, no traces of it to be found after forty-eight hours, or at the most, only a little bloody serosity. Supposing that the effusion amounted to 500 grammes only a small clot will be found at the end of three days: more than four-fifths of the fluid will have been absorbed.

Even when the experiments were made with from one to three kilogrammes of blood, more than half of the entire quantity was found to have disappeared in forty-eight hours: after three days, there only remained, as in the former case, a small clot and a little reddish serosity. Throughout our experiments, we did not in a single instance find the slightest trace of inflammation of the pleura. I grant that, perhaps, the pleura of a horse is more tolerant than the pleura of a man: I admit also that, perhaps, the blood may be the temporary cause of inflammatory determination to the pleura; but still, from the cases and experiments which I have laid before you, am I not entitled to say that in cases of traumatic extravasation into the pleura, the surgeon ought to remain as a spectator? Absolute rest, and very low diet, were probably the best means of promoting absorption.

Wounds of the chest complicated with pleural hæmorrhage are,

however, sometimes, frightfully dangerous, irrespective of the loss of blood, a fact which our experiments do not fully explain. I must therefore, add a few words to the remarks I have already made on this subject.

The blood injected into the pleura does not there comport itself after the manner of a foreign body. It does not seem to irritate the serous membrane more than food irritates the stomach, than fæcal matter irritates the colon, or than urine irritates the bladder. But we know that sometimes the urine does irritate the bladder: it does so when there is a change in its character. Affections of the bladder also often occasion changes in the urine; but on the other hand, an altered state of the urine may cause catarrh of the bladder. The case is similar in respect of blood extravasated into the pleura. If the wound of the chest give rise to an escape of air as well as of blood into the pleura, there will be an immediate change in the character of the blood, which will then act as a foreign body. When, in our experiments, we allowed the blood to accumulate in an evaporating vessel, and when some hours later, we introduced the coagulated blood into the pleural cavity, it putrefied there; and the animals sunk under formidable attacks of pleurisy. This experiment is an additional proof of the dangers which attend the operation for empyema, when performed with a view to remove coagulation from the chest.

But if, in spite of the physician, blood and air make their appearance simultaneously in the pleural cavity, violent inflammation is kindled: it is then a duty to resort, with the least possible delay, to the operation for empyema—to inject the tincture of iodine. In a word, it is necessary to act in the same way that I have counselled you to proceed in formidable collections of pus, and in hydro-pneumothorax.

LECTURE XXXIV.

HYDATIDS OF THE LUNG.

Hydatids of the Lung though rare are not so rare as Hydatids of the Pleura.— Diagnosis is exceedingly difficult.—Resemblance to Pulmonary Phthisis.—Possibility of Cure by Spontaneous Evacuation by the Bronchial Tubes.—Reserve required both in respect of the Prognosis and Treatment.

Gentlemen:—The examples of hydatids of the lung given by Dr. Davaine in his beautiful work on the entozoa, are comparatively very few:[1] and if you make inquiries on the subject of your hospital teachers, you will find most of them admitting that they have never met with a case of this affection. Bricheteau, who specially devoted his attention to diseases of the chest, only saw two cases of it during a medical practice of more than forty years; and my honourable and learned colleague, Dr. Andral, has only recorded five cases. Professor Monneret has only met with a single instance, and that was detected on the dead body. For my part, I had likewise only seen a single case, till I met with that which I now propose to make the text of some remarks on this singular affection.

You will recollect that the patient to whom I refer was a young man of seventeen years of age, who, about the end of December 1861, became a patient in St. Agnes's ward. On his admission, I found that he had acute general bronchitis, and that the right lung was most affected. From hearing on that side coarse mucous râles like gurgling, a prolonged expiratory sound, diminished resonance on percussion over the infra-spinous fossa of the scapula, and finally hippocratic deformity of the fingers, I was led to fear that the bronchitis was only symptomatic of tubercles. This hypothesis was all

[1] Davaine:—Traité des Entozoaires de l'homme et des animaux domestiques. 8vo. Paris: 1860.

the more probable from the fact, that the patient was said to have been liable to take catarrhal affections every winter from the time he was six years old; and that it was added he had had, on different occasions, profuse hæmoptysis. I nevertheless reserved my diagnosis, the acute catarrhal affection of the bronchial tubes masking the characteristic signs of the tuberculous affection. The acute symptoms having moderated, the fever having ceased, and the râles steadily diminishing, the respiratory sound in the right lung seemed to me to become more normal. Some days later, however, there was a return of the fever, particularly in the evening; and the young man complained of pains in his right side. On examining that side of the chest we found dulness on percussion in its two inferior thirds, absence of thoracic vibrations, and the presence of broncho-egophony; these omens which conjoined with marked oppression in the breathing, indicated the existence of pleuritic effusion complicated with bronchitis, and anew characterised by mucous râles, and muco-purulent expectoration. These local symptoms and the bad general state of the patient, led me to fear that new tubercular mischief was going on in the right lung, when all at once during the night between 18th and 19th January, he was seized with great difficulty of breathing, accompanied by a threatening of suffocation, and after some violent paroxysms of cough, he ejected by the mouth a great quantity of muco-purulent matter. This afforded him some transient relief: but renewed attacks of coughing induced renewed purulent vomiting. Next morning, I ascertained that the quantity which he had brought up was half a litre. The vomiting now described was followed by a great change in the symptoms. I thought that the effusions, the existence of which I had detected on the previous evening, had found an exit by an opening in a bronchial tube, and as there was no sign of hydro-pneumothorax, I supposed that there was very probably an encysted or interlobular pleurisy, because on attentively examining the contents of the spittoon, whitish shreds of false membrane were seen. When these shreds were carefully washed, they appeared white, opaque, thickish, torn at the edges. Notwithstanding the great rarity of such cases, I came to the conclusion that what we saw was the *débris* of an hydatid tumour of the lung. This view was established, beyond any doubt, to be correct by a microscopical examination made by M. Charles Robin. For three days, the patient continued to eject fragments of false membrane, and muco-purulent matter mixed with a little blood. The expector-

ation gradually diminished in quantity, the fever ceased, and day by day, there was an appreciable amelioration in the state of the patient. Very soon there was no dulness on percussion in the inferior and posterior part of the lung, expiration continuing, however, to be blowing in that situation, where coarse mucous râles were still audible. Convalescence advanced rapidly; and, all the local phenomena having disappeared, the young man left us quite cured, after two months' residence in hospital.

The principal points in this interesting case may be thus summed up:—The rational signs were those of pulmonary phthisis: the physical signs were doubtful: there was bronchitis and pleurisy: then, under the influence of the acute disease, a hydatid tumour, developed in the inflamed lung, became the seat of inflammation, an eliminative process began around it, and the patient ejected hydatid fragments mixed with a vast quantity of pus. Immediately consequent upon the ejection of a great quantity of muco-purulent matter, auscultation revealed the existence of a cavity in the inferior third of the right lung, where blowing respiration could be heard. By degrees, this cavity disappeared, the normal respiratory murmur again became audible throughout the whole extent of the chest; and the general state of the patient became more and more satisfactory. I said that in all probability the hydatid had its seat in the lung, because after the vomiting we could discover no signs of hydro-pneumothorax, which signs would certainly have existed had the tumour projected into the pleural cavity.

Gentlemen, before commenting on this case, which is similar to others, and suggests considerations relative to the difficulties attending the diagnosis of hydatids of the lung, the progress of the case, and the different modes of termination, let me, in passing, direct your attention to the semeiology of that deformity of the fingers which we have observed in our patient.

It is stated in the works of Hippocrates that the nails of phthisical subjects become contracted—*tabidis ungues contrahuntur*—and crooked—*tabidis ungues adunci*. This clinical fact, though not denied, was forgotten, till 1832, when Dr. Pigeaux pointed it out anew. In the following year, I published in the *Journal des Connaissances Médico-Chirurgicales* a paper on this subject, accompanied by a plate drawn by my pupil Dr. Jardon; and now there is no physician ignorant of what is meant by the expression—"hippocratic deformity of the fingers." This deformity consists in contraction of

the ungual phalanx, with enlargement and thickening of the digital pulp. Whilst the nail becomes curved towards the palm of the hand, the extremity of the finger assumes the form of the large end of a club, and sometimes in enlarging, it flattens so as to resemble the head of a serpent. This deformity, generally, comes on by slow degrees; but at other times, it is produced with great rapidity, the patients suffering pain from the change which is going on. The other phalanges do not undergo any change. In some persons, the toes are the seat of a similar deformity; but when it occurs in the toes, it is generally in a much less degree than in the fingers.

Hippocratic deformity of the fingers is chiefly observed in persons who have reached the second or third stage of pulmonary phthisis: it is not met with in scrofulous subjects, and it seldom exists in patients affected with abdominal phthisis, unless they are likewise the subjects of pulmonary tubercle.

It is also observed, as the older physicians stated, in individuals affected with non-tuberculous chronic diseases of the chest. Some years ago, I observed it in a child whom I believed to be tuberculous, and in whom paracentesis of the chest, performed for an enormous pleuritic effusion, had left a fistula, by which for several months a large quantity of purulent serosity was discharged. This child grew up to adolescence, retaining the fistula: the chest had undergone considerable contraction, but I was never able to detect any signs of tubercle. In 1859, I had a female patient, whom I twice tapped—the interval between the tappings being short—in a case of empyema following parturition, who retained a thoracic fistula for two years: this woman had hippocratic deformity of the fingers, but I could not detect in her any sign of tubercle. I believe, then, that hippocratic deformity of the fingers may be an accompaniment of chronic chest affections unconnected with phthisis. The two cases which I have narrated, and others which I could cite, show that it is liable to occur in diseases of the pleura: I have observed it in patients with bronchitis, with emphysema, and in others who had nothing more than nervous asthma: I have also seen it in patients with organic disease of the heart. It must be remembered, however, that it is principally in cases of phthisis that it is met with, and that the curving of the nail is the more marked, the more advanced is the stage of phthisical disease. For this reason, hippocratic deformity of the fingers has some value as a diagnostic sign of phthisis.

Gentlemen, excuse me for making this digression. In clinical

studies, facts apparently the most insignificant may have their importance; and so we ought not to neglect them.

Let us now return to our subject—*hydatids of the lung.*

The details into which I entered in reference to our patient of St. Agnes's ward, have shown you the embarrassing nature of the diagnosis. The difficulty lies in the fact, that there is no special sign of hydatids of the lung. Study the cases which are recorded in scientific works, and you will perceive that in a great number, perhaps in the majority of them, the morbid phenomena are sometimes dependent on pleuritic effusion, and sometimes upon pulmonary phthisis. Of course, when the hydatids, or fragments of hydatids, have been expectorated, there is no room for doubt as to the nature of the case; but there will still be an uncertainty as to the precise seat of the tumour: it will be a question whether it is situated in the parenchyma of the lung or in the pleural cavity, or whether the hydatids have not come from the liver by way of the lungs.

Intra-thoracic hydatid tumours occur much more frequently in the parenchyma of the lung than in the pleural cavity, as has been shown by M. Davaine by an analysis of cases collected by him. This opinion had been previously enunciated by Laennec, and is likewise that of Professor J. Cruveilhier. By simply reasoning from analogy, moreover, the same conclusion might have been come to; for it is in parenchymatous organs, such as the liver, spleen, and kidneys, and in the thickness of muscular masses, that hydatids are generally, or indeed nearly always, developed. The respiratory apparatus is no exception to this general rule: and M. Davaine believes that a great many alleged hydatids of the pleura, are really hydatids of the lung which have fallen from their original situation into the cavity of the pleura. It may likewise happen, that tumours situated near the periphery of the lung, as they slowly develope themselves at the edge of that organ, may become detached, to a greater or less extent, from the pulmonary pleura, which they push over to the costal pleura, in such a manner that the hydatid pouch seems to be placed in the serous cavity, although in reality it is wholly external to it. Such seems to have been the state of matters in a case reported by Dupuytren and Geoffroy, and designated by them—*double cyst of the pleura.* In that case, it is stated, that the patient had had numerous attacks of hæmoptysis, which one can hardly understand, unless the cyst had in the first instance occupied the pulmonary parenchyma, as we know that hæmoptysis is a very common symptom of diseases

of the lung, while it never supervenes in affections of the pleura.

Hæmoptysis, moreover, has been remarked to have occurred in nearly all the cases of pulmonary hydatids. A man whose case has been published in the *Bulletins de la Société Anatomique* by M. Husson, expectorated hydatids upon fifteen different occasions, and each time the occurrence was preceded by spitting of blood. He never had any of the rational or physical signs of pulmonary tuberculisation; and his general health was satisfactory.

Gentlemen, when hydatid tumours seated near the periphery of the pulmonary parenchyma are slowly developed in the direction of the pleura, there can be no symptoms except those which accompany more or less pleuritic effusion, or are caused by the squeezing of the lung into the vertebral hollow. You can understand, however, that these symptoms are more or less serious, and that when they are caused by a double cyst, as in the case narrated by Dupuytren and Geoffroy, the embarrassment in the breathing may proceed to such an extent that the patients are carried off by suffocative fits.

But when a hydatid tumour of the lung bursts suddenly into the pleural cavity, the symptoms are very much more serious, as a subacute pleurisy is set up, and when the bursting is both into the pleura and the bronchial tubes, there is produced hydro-pneumothorax, as happened in the following case, recorded by Dr. Mercier in the *Bulletins de la Société Anatomique*. A man, thirty-eight years of age, subject for years to frequent hæmoptysis, although he presented no other sign of tubercular disease at the summit of the lung, was suddenly seized with acute pain in the right side: on examining the chest, hydro-pneumothorax was discovered: the patient sank rapidly. At the autopsy, there was found in the pleural cavity, a hydatid floating in the effused fluid: in the part of the lobe of the lung corresponding to the interlobular cleft, there was an excavation in the parenchyma of the organ, and in the same situation was observed an ulcerated bronchial tube.

It is evident, or at least very probable, that in this case, the process of elimination going on simultaneously in the bronchial tubes and pleura, led to perforation of the lung, and so to hydro-pneumothorax. The attacks of hæmoptysis which occurred during life, the discovery after death of an excavation in the parenchyma of the lung still containing the hydatid, seemed to point

out clearly the seat of the affection. However, inasmuch as the excavation was situated in the interlobular fissure, it may be asked whether the pouch was not originally formed in that fissure, whence it had invaded and excavated the pulmonary parenchyma: and on comparing this case with cases in which hydatids unquestionably occupied the lung itself—in considering, as I have just been saying, that it is generally in parenchymatous organs that these entozoa become developed, it was justifiable, arguing from the general to the particular, to conclude, that in this case the original seat of the tumour was really that which the examination of the dead body enabled us to assign to it.

This case may give you an idea of the difficulty which there is sometimes of determining in the dead body the precise seat of an hydatid of the lung, when the tumour, not being situated in the interior of the parenchyma, has burst at the surface of the organ. I need not say that the difficulty will be infinitely greater when we make our examination in a living patient. However, when the radical cure takes place, after the ejection of hydatids by expectoration, the physician may conclude that the hydatids occupied the substance of the lung, the nature of the affection being shown by the discharge from the bronchial tubes; and when a hydro-pneumothorax is produced, it may be presumed, not only that the hydatids are situated in the pleural cavity, but also, that they occupy a position near the periphery of the lung.

Sometimes, the hydatids are not enclosed in an adventitious cyst; and at other times, the containing cysts are exceedingly thin, a circumstance which well explains the facility with which they may find an exit by the bronchial tubes, when these canals are opened by ulceration. This was pointed out by M. Houel in a report which he read to the Anatomical Society upon the occasion of M. Pinault communicating a case of pulmonary hydatids. The absence of the adventitious cyst, or the extreme tenuity of the envelope by which the cyst is constituted, likewise explains how hydatid tumours of the lung may become ruptured under the influence of an inflammatory affection of the respiratory apparatus, as occurred in the case of our patient of St. Agnes's ward.

Gentlemen, from M. Davaine's statistical researches, it appears that it is much more unusual to meet with several hydatid tumours in a single lung, than a single hydatid tumour in each lung; but it is still more common to find a single hydatid in only one lung, and in that

case, it is generally in the right lung—usually in the inferior, but sometimes in the superior, lobe.

The much greater frequency of hydatids in the inferior lobe of the right lung, considered in connection with the extreme frequency of these entozoa in the liver, has led to the supposition that, in a certain number of cases, the intra-thoracic hydatids have passed from the liver into the chest.

There have now been recorded numerous cases in which this passage of hydatids from the liver into the thorax has taken place. In 1856, Dr. Dolbeau called the attention of physicians to the tendency which[1] large cysts of the convex surface of the liver have thus to invade the chest, pressing the diaphragm against the lung, at the same time depressing the liver, and so gaining the epigastric region. The invasion of the thoracic cavity by these cysts may be so great, that the lung, packed into the clavicular region, and into the vertebral hollow, is reduced to a third, or even, it may be, to a fourth of its normal volume. You can understand how such an invasion of the chest by an abdominal tumour, cannot take place without the diaphragm becoming exceedingly attenuated. This is what takes place:—the attenuated diaphgram contracts adhesions with the hydatid pouch, which is consequently dragged up in the ascending movement. The result of this is, that when it it is wished to determine the nature of the affection with which we have to do, we may possibly find nothing more than signs of a pleuritic effusion—absolute dulness of a greater or less extent of the lower part of the chest, absence of thoracic vibration, absence of vesicular murmur, absence of blowing and egophony, the results of the displacement of organs, and crushing up of the lung, the place of which is occupied by a liquid tumour. The progress of the disease, and the thoracic deformity (a deformity which extends to the region of the liver, where it presents a peculiarly characteristic aspect), furnish the only elements of diagnosis.

The slow unobtrusive inflammation which has caused the formation of the adhesions between the hepatic cyst and the diaphragm, may, extending, in virtue of contiguity, to the pleura and the lung, produce similar adhesions between the lung, the pleura, the diaphragm, and the tumour, which adhesions conduce to the favorable termination of the disease. If—as occurs in a few exceptional cases—adhesions do not form between the hydatid pouch and the lung,

[1] Dolbeau:—Etudes sur les grands Kystes de la surface convexe du Foie. *Thèse de Paris*, 1856.

the pouch opens, through a perforation of the diaphragm, into the pleural cavity, occasioning an almost invariably mortal pleurisy. But if the adhesions are such that the lung, the pleura, the diaphragm, and the cyst, are intimately united, the tumour, which ultimately always bursts, opens into the cavity it has dug for itself in the pulmonary parenchyma, and discharges by the bronchial tubes.

A great number of cases of this character have now been published; and, among other places, you will find them in the thesis of Dr. Cadet-Gassicourt,[1] and in the memoir of Dr. E. Leudet (of Rouen).[2] Bricheteau setting forth all the interest which attaches to this subject, pointed out the propitious manner of evacuating hydatid tumours by the bronchial tubes.[3] In the case of a patient whom he saw with Professor Natalis, Guillot states, that immediately after the patient had expectorated matter containing the débris of hydatids, there was detected, by auscultation, a cavity excavated both in the pulmonary parenchyma and in the liver; and which was characteristically indicated by amphoric blowing and pectoriloquy. That the liver was the seat of the tumour was sufficiently shown by the expectoration being constituted by a yellow fluid which assumed the colour of verdigris when treated by nitric acid. At the time of bringing it up the patient felt a very marked saline taste in the mouth, due probably to the chloride of sodium, which chemical analysis has shown to exist in hydatid cysts.

This fluid, taking a yellow colour from the bile, has also sometimes a chocolate-brown hue derived from the colouring matter of the blood, and also from microscopic hepatic cells. These facts which have served as the basis of Bricheteau's work—a work to which I refer you—were obtained in his hospital practice, and he has added to them others, quoted from the curious memoir of Hébréard, formerly a physician of the Bicêtre. But what I have to say on this subject, will be more in place when I come to speak of cysts of the liver: I therefore defer to another occasion some remarks required to complete my account of this subject. I shall only add

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[1] Cadet-Gassicourt:—Recherches sur la Rupture des Kystes Hydatiques du Foie à travers la paroi abdominale et dans les organes voisins. *Thèse de doctorat*, Paris, 1856

[2] Leudet:—Mémoire sur le Traitement des Kystes Hydatiques du Foie, lu à la Société Médicale des Hôpitaux. [*Archives Générales de Médecine*, for January and February, 1860.]

[3] Bricheteau:—in the *Revue Médico-Chirurgicale* for 1852.

that the thesis of Dr. Cadet-Gassicourt has given us new means of diagnosing intra-thoracic cysts, a very important matter for clinical physicians.

Before concluding, I must succinctly mention a case published by Dr. Vigla.[1]

The patient was a man, aged thirty-two years, who, consequent upon a violent contusion produced by the kick of a bull on the right side of the chest, complained of pain in the right hypochondrium, and an oppression of the breathing, which from the date of the accident, fifteen months previously, had been constantly increasing. For the preceding five months, the dyspnœa had been so considerable, that the patient had been obliged to give up his employment. He had little or no cough, and no expectoration: he had never had hæmoptysis: although he had in a marked manner the symptoms of anæmia, he complained of no organic suffering, except that of which the respiratory organs seemed to be the seat; and he stated that the pulmonary symptoms had never been accompanied by fever.

The intense pain of which he complained seemed to be limited to a small space under the right mamma. The oppression of the breathing, which was continuous, became excessive when he walked, or even after the exertion of speaking for some time: he could not lie on the left side, and generally sat when in bed. On examining the chest, it was observed that there was a much greater development of the right than of the left side, and that anteriorly the right side was very much arched in appearance: there was distension of the intercostal spaces, which projected at least as much as the ribs. On the right side also, the normal resonance was completly replaced by absolute dulness, which extended from the second intercostal space to the umbilicus, measuring—taking a line parallel to the sternum—28 centimeters, and —transversely—crossing the median line in such a manner that the space occupied by it was circumscribed below by a line, which after passing beyond the navel, proceeded in an oblique direction under the left axilla; and above, by a line which, following the upper edge of the second rib, passed over the sternum at three centimeters below the bifurcation of that bone, and proceeded by a curve to rejoin the lower line under the left axilla. Thus, it occupied the whole of the

[1] Vigla:—Mémoire sur les Hydatides de la cavité thoracique. [*Archives Générales de Médecine*, for September and November, 1855.—Vol. II of fifth series.]

right side of the chest, encroaching a little on the left side. Applying the hand to this side, and at the same time requesting the patient to speak, it was ascertained that an entire absence of thoracic vibration existed; and on applying the ear, there was heard neither vesicular murmur, nor any anomalous sound in front, although posteriorly, the respiratory sound was exaggerated in the three superior fourths of the right side, as it was also in the left side. There was heard, moreover, on the right side, amphoric resonance of the voice, and even respiratory murmur, like that heard in certain pleuritic effusions, unaccompanied by blowing or egophony.

No lesion of the pulmonary parenchyma, as is justly observed by Dr. Vigla, seems capable of producing similar deformity of the chest. Nor was there any ground for entertaining the idea that there was hydrothorax; for it would be difficult to believe that an encysted pleuritic effusion could be distributed so unequally and so irregularly as to respect the first intercostal space, the three superior and posterior fourths of the right side of the thoracic cavity, while it invaded the left side, and pushed the diaphragm as far down as the umbilicus. The hypothesis that there existed a solid tumour—a cancer, an aneurism of the aorta or of one of its principal branches—was untenable. In the first place, a solid tumour would transmit the respiratory and cardiac sounds, which were entirely absent in this case; and then again, a cancerous tumour, the only tumour which could take so large a development, could not have been formed without producing general cachectic symptoms. Dull and deep-seated fluctuation was perceptible, and furnished a sign, which, in conjunction with others, justified the presumption that there was a hydatid cyst.

An exploratory puncture made by M. Monod, surgeon to the Maison de Santé, proved this diagnosis to be correct. The fluid which issued from the capillary canula of the trocar was clear as water from the crystal spring: it produced no change on litmus paper, and no albuminous precipitate when treated by nitric acid or heat. Tapping, consequently, was performed with a larger trocar, when 2,450 grammes of fluid similar to that originally withdrawn were evacuated. That portion of this fluid which flowed last from the canula, contained the shreds of transparent membrane, which M. Charles Robin found on examination to be the débris of hydatids.

A solution of iodine was injected. Thirty-seven days after the operation—fifty one days after his entering the Maison de Santé—the patient asked and received permission to leave, that he might

resume his employment. Eleven months afterwards, when M. Vigla saw him, the cure was as complete as possible.

Gentlemen, let me say a word to you on the great value of that *peculiar arching of the thorax*, observed in the case of which I have just given you an abridged history. It is a diagnostic sign full of meaning, and is in itself sufficient to justify an exploratory puncture such as M. Vigla practised in his case. This form of arching of the chest, so very peculiar, enabled me so far back as 1848, to diagnose an intra-thoracic hydatid tumour in a girl seven years old.

This little girl presented the general appearance of a phthisical subject. For a long time, she had had cough and oppression of the breathing. Her emaciation was extreme. On examining the chest, I found complete dulness on percussion, and an absence of thoracic vibrations: there was a globular projection of the thorax, the maximum of which corresponded with the sixth and seventh ribs. As there had been no hæmorrhage, and as auscultation did not disclose any lesion of the upper parts of the lungs, I proposed to tap the chest. I was not allowed to do so: and the child died some weeks after my visit.

Gentlemen, the clinical history of hydatid cysts of the lung is far from being complete. The insidious commencement, the sometimes slow, and at other times rapid progress of the affection, our almost complete ignorance of its etiology, sufficiently proclaim the difficulty of the subject. In the majority of cases, the nature of the affection has been mistaken, and very rarely even suspected during the life of the patients, so that for a long period the only data which we had in relation to it were those furnished by pathological anatomy. These data, however, were important, and such as to throw light on the manner of forming a diagnosis on the living subject. They informed the physician, for example, that he might meet with hydatids in the lung, but that they seldom if ever existed in the pleura they told him that hydatids of the liver might pass from the liver into the thoracic cavity: that the existence of hydatids in the chest generally coincided with their presence in other organs, particularly in the liver, and that their favorite locality is the right lung.

Pathological anatomy has also taught us that pulmonary hydatids, of which generally there is only one found in the same lung, may, within the parenchyma of the lung, become as large as the head of an adult; that their adventitious envelope may either be very thin or entirely wanting; that an acute inflammation of the lung may cause

them to burst, either into the pleural cavity where they produce the symptoms of hydrothorax, or into the bronchial tubes, in which case they may be expectorated either in shreds, or in their totality. Pathological anatomy has shown us that these vast pulmonary and hepatic warrens communicate by one large diaphragmatic fistula. In these facts, there is enough to lead us to suspect in some cases, and to affirm in others, that our patients have pulmonary hydatids. An attentive examination of the other organs, and the progress of the affection, ought to enable us to bring together all the probabilities in such a manner as to determine the original seat of the affection which we suppose to exist.

To Hébréard and Brichetcau, and to MM. Vigla, Cadet-Gassicourt, and Davaine, belong a large share of the merit, which I am pleased to acknowledge, of elucidating this important question of the diagnosis of hydatids of the lung.

We may suspect the existence of hydatids of the lung, if, along with the co-existence of certain symptoms, we find that peculiar deformity of the chest of which I have been speaking. When once the existence of the affection has been made out, it is then necessary to try to determine which is the probable original seat of the entozoa.

In general, patients affected with hydatids of the lung, present many of the rational and physical signs of phthisis or chronic pleurisy. In fact, the majority of this class of patients will tell you that they have been subject for a long period to hæmoptysis, more or less profuse, and more or less frequent, as well as to oppression of the breathing. You will hear râles disseminated over the chest, and sometimes you will find dulness at one or both summits, when tubercles will coexist with the hydatids. But independently of this exceptional complication, the attentive study of the progress of the affection, the rational explanation of some of the symptoms, will enable you to reject the hypothesis that there is tubercular phthisis, when, for example, there is no disease of the summits, as in the case observed by M. Husson: the hæmoptysis in such circumstances will then probably have no other cause than the continuous irritation excited by the presence of a foreign body, which will frequently occupy the middle, and still more commonly the lower, lobe of the lung. The general condition of the patient, his age, and the progress of the affection, will aid your diagnosis; and if you have had occasion to suspect the presence of hydatids in the liver, or in any

other organ, you will be entitled to conclude that the pulmonary parenchyma is itself their seat.

Again, attention is not directed to hydatids of the lung till they have attained a great size, when they are liable to be mistaken for several descriptions of encysted and interlobular pleurisy, as occurred in the case of the young man, our patient of St. Agnes's ward. However, when, on examining a patient, you find a globular deformity of the chest of limited extent, the probability of the case being one of encysted hydatid is greatly strengthened: sooner or later, the progress of the affection, which is very different from that of pleurisy, and particularly the expectoration of hydatids, when it occurs, will remove all doubts. You will perhaps be justified, even in cases in which this valuable diagnostic occurrence does not take place, to suppose that there are hydatids, when you see sudden symptoms of inflammation of the pleura, while at the same time there is flattening of the globular tumour, because there will then be ground to suppose that the flattening is consequent upon rupture of the hydatid pouch into the pleural cavity. This probably correct diagnosis will attain almost a certainty of being correct, if symptoms of acute pleurisy come to be added to the signs of hydro-pneumothorax. In such a case, ulceration, in virtue of which the hydatid has emerged into the pleura, has likewise, at the same time implicated a bronchial tube.

I have said that it is needless to insist on the fact, so palpably evident, that when the débris of hydatids has been discovered in the expectoration, there can be no possible doubt as to the nature of the case. There will still remain, however, some uncertainty as to the original seat of the hydatids. This is a point in diagnosis which has to be elucidated.

Beyond all doubt, hydatid cysts have been found in the pleural cavity. M. Vigla's case, two similar cases narrated in his paper, furnish additional proof of this, up to a certain point. As for myself, I accept, with everybody else, the existence of these pleural cysts. But if an attentive analysis and a careful study be made of the cases, there will be found reason to believe that hydatids of the lung, which have fallen into the pleural cavity, have often been mistaken for pleural hydatids. This, you remember, occurred in the case reported by Dupuytren and Geoffroy. Even M. Vigla's case is very open to discussion from this point of view, because as the patient fortunately recovered, there was no necroscopic examination

to verify the diagnosis. Besides, if we consider, as I remarked at the beginning of this lecture, that hydatids generally develop themselves in parenchymatous organs—the liver, spleen, kidneys, and ovaries—we are led to conclude that the lungs form no exception to the general law, and that acephalocysts are much more frequently met with in the lungs, than in the pleura. Dr. Davaine, whom every one admits to be an authority on this subject, is quite convinced that hydatids of the pleura are very rare. The result of his laborious researches is to the effect, that in twenty-five cases of hydatids which he examined, there was only one in which evidence existed of the primitive development of the hydatid having been in the pleural cavity.

The diagnosis of the precise seat of intra-pleural hydatid cysts is rendered all the more difficult by the circumstance, that hydatids of the convex surface of the liver may invade the chest, either pushing up the diaphragm without perforating it; or, without bursting, they may make a passage for themselves through the distended attenuated fibres of that muscle. It may be asked, if this be not what happened in M. Vigla's case—whether his case be not similar to that reported by Professor J. Cruveilhier[1] in which a hydatid cyst of the liver, which had penetrated into the pleural cavity, was evacuated by paracentesis of the chest, a cure taking place as in M. Vigla's patient. I am aware, however, as I have been careful to tell you, that cases of this description are of exceedingly rare occurrence: that very commonly, hydatids of the liver rupture, and thereby cause a rapidly mortal pleurisy; or, as is still more usual, adhesions form between the diaphragm, pleura, and lung, so that when the hydatids open into the cavity which they have formed by burrowing in the pulmonary parenchyma, they empty themselves into the bronchial tubes.

When the latter occurrence takes place, just as in that which I pointed out as happening in hydatids of the lung, the elements for forming an opinion possess an almost absolute degree of certainty. Apart from the circumstance of fragments of hydatids, or entire hydatids, being found mixed up with the expectorated matter, that matter presents peculiar and unmistakable characters. In it we find a yellow, thick, oily fluid, which, on the addition of nitric acid,

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[1] Cruveilhier:—Dictionnaire de Médecine et de Chirurgie en 15 volumes:—article, Acephalocystes: Paris, 1829.

assumes the colour of verdigris, evidently dependent on the presence of the colouring principles of the bile. The fluid has sometimes a chocolate-brown colour, due to an admixture of a certain quantity of blood. Then, again, there is a diminution of the tumour in the right hypochondrium caused by the increased bulk of the liver; and the hitherto impeded movements of the diaphragm become more easy. Finally, gurgling, amphoric blowing, and vocal resonance heard on applying the ear or the stethoscope to the situation formerly occupied by the tumour, show that a cavity exists which is evidently excavated partly in the lung and partly in the liver.

Gentlemen, when you have diagnosed an intra-thoracic hydatid, be very reserved as to the prognosis. While you have a right to hope that all may go on favorably, that the malady may reach a happy issue by the unaided efforts of nature, and through the mechanism which I have explained to you at such length, you must not forget that the work of elimination, though in itself favorable, is not unattended by danger. At its commencement, it may occasion suffocative paroxysms: the presence in the air-passages of the hydatids and of fluids which irritate the larynx, the trachea, and the bronchial mucous membrane, may occasion fits of coughing, which in their turn may lead to mortal hæmorrhages, an occurrence of which an example has been reported by Dr. Pillon. You have to dread hydropneumothorax, and its disastrous consequences. You have also to dread asphyxia, a consequence of respiration being interfered with by the tumour attaining so large a size as to compress the lung: this happens not only in cases like that of Dupuytren and Geoffroy, in which the intra-thoracic tumour was double, but likewise when there is only an hydatid on one side.

When these unfavorable circumstances do not exist, and when the hydatids find their way out of the body by the bronchial tubes, you may not only hope for recovery, but also for an early cure. The inflammatory symptoms which attend the process of elimination, cease, the fever subsides, the appetite returns, and at the end of some weeks, there may be perhaps a complete restoration to health.

Ought there to be any active interference on the part of the physician with intra-thoracic hydatids? It is most prudent to abstain from such interference. Here, as in many other circumstances, we must know how to wait, while we attentively watch the patient, seeking to moderate inflammation and sustain the vital powers.

So far am I disposed to go in advising extreme caution, that I

even recommend you to abstain from exploratory punctures, which there is a temptation to make, with a view to elucidate the uncertainties of the diagnosis. These punctures may prove fatal, if adhesions have not been formed between the tumour and the walls of the chest, by causing effusion into the pleura, the dangers of which occurrence I have pointed out to you. Now, it is impossible for the most experienced physician to affirm that such adhesions exist. When circumstances peremptorily compel you to interfere so as to give exit to the fluids, the first indication is to excite adhesive inflammation, the existence of which is absolutely necessary: this, however, cannot be brought about, unless the tumour is in contact with the thoracic walls, and unless there is no intervening portion of lung. This indication will be fulfilled by making numerous acupunctures, and repeating them on several successive days.

When adhesion has taken place, the pouch may be emptied by tapping it with a bistoury or large trocar, after which it has to be injected with a solution of iodine.

This is a mode of treatment exactly similar to that which I adopt for the cure of hydatid cysts of the liver, a subject upon which I hold myself in reserve till an opportunity occur, when I shall fully go into it. I would now only add in conclusion, that I have never practised this method for the cure of hydatids of the lung, that I do not know whether it ever has been so employed; and consequently I cannot say what might be the results of having recourse to it.

LECTURE XXXV.

PULMONARY ABSCESSES AND PERIPNEUMONIC VOMICÆ.

Rare Affections, if we exclude from the category Tubercular Vomicæ and Metastatic Abscesses.—Most frequent in Children, in whom they are the result of Lobular Pneumonia.—Diagnosis of Peripneumonic Vomicæ is Difficult.—They may be confounded with Pleural Abscesses.

Gentlemen :—At the close of my last lecture, I showed you the lungs of two patients who died in our wards of acute pneumonia. At one of the autopsies from which these lungs were obtained, you may have seen that an immense pouch of pus occupied the anterior and lower portions of the superior lobe of the left lung. This cavity was large enough to hold a large egg of a hen: it was divided by incomplete partitions into chambers communicating with one another: the walls of the cavity were formed by grey indurated pulmonary parenchyma. The cavity communicated with the pleural cavity by a large opening, shaped like a button-hole, situated at the anterior margin of this pulmonary lobe, and probably measuring about two centimeters in length. In every other situation, the parenchyma of the lung seemed to be healthy, and to present no trace of tubercle. In the cavity, there was no trace of anything like tubercular matter: and there was not the slightest exhalation of a gangrenous odour. The corresponding pleural cavity was filled with a great quantity of creamy, inodorous, white pus. The surface of the visceral and parietal serous membrane was covered, in the two lower thirds with a layer of matter, pultaceous, pseudo-membranous, thick, and of a greenish white colour. The lung adhered closely to the walls of the chest along the vertebral column, as far down as the diaphragm; but on dragging the adhesions, they easily gave way, except near the diaphragm, where they resisted so much that it was necessary to remove the

diaphragm with the lung. The left lung, one third less voluminous than the right, had its superior lobe flattened upon itself, and applied along the spine. An attempt to inflate it failed, in consequence of the air escaping by the opening into the cavity of which I have spoken.

There was nothing abnormal in the condition of the right lung, excepting some old adhesions, which did not offer much resistance.

At the autopsy of the other case, you also saw a large purulent cavity in the left lung, but it was in a less advanced state, and in fact was only beginning to form. It was, moreover, the result of a circumscribed or partial peripneumonia, whereas in the other case, it existed in the midst of a lobe inflamed throughout its entire extent.

The pulmonary tissue presented, in fact, the consistence of hepatic tissue. The two lobes of the left lung were completely involved, and had a very manifest grey colour: when an incision was made in the condensed parenchyma of the lung, a great quantity of a frothy greyish sanies oozed out from the incised surface. The tissue was easily torn by the pressure of the finger: at the superior and posterior part of the inferior lobe, was situated the purulent cavity of which I have been speaking. It was as large as the abscess we met with in our first autopsy: it was quite full of putrilaginous matter of a lateritious appearance, and only separated from the interlobular fissure by a very thin plate of pulmonary tissue. The examination was made with great care, and without any violence, so that it did not seem probable that there had been any accidental breaking up of tissue by the pressure of the hand of the pupil who made the necropsy: still, I must tell you that I have my doubts on this point.

These two cases, Gentlemen, are examples of what have been called *vomicæ*—abscesses of the lung: they are phlegmonous abscesses, very different from the purulent collections met with in tuberculous subjects, very different also from the abscesses termed *metastatic*, which we meet with in the bodies of persons who have been carried off by purulent infection, and which are characteristic of the purulent diathesis.

These non-tuberculous, non-metastatic, purely inflammatory vomicæ are very rare lesions *in adults*. I make this limitation, because in young children they occur very frequently. On this point, I fully concur with recent clinical observers who have written on the pneumonia of children. In a certain restricted sense, however, the general rule is likewise applicable to children; for it is only in

lobular pneumonia that pulmonary abscesses are met with, and *lobular* is a very different affection from *lobar* pneumonia.

Pulmonary abscesses in children sometimes occur disseminated in very small number throughout the pulmonary parenchyma: at other times, they are placed so closely together as to resemble myriads of tubercles. They are met with least frequently under the latter aspect. When they are very few in number, they either form small pouches on the surface of the lung, producing prominences under the pleura; or, having emptied themselves by the bronchial tubes, they contain only air; or possibly, they may contain a mixture of air and pus. In these different states, it is difficult to say whether the pouch has been formed by a suppurating lobule, or by dilatation of the extremity of a bronchial tube terminating in a lobule, the cells of which have been ruptured: in the latter case, it would be merely a variety of vesicular emphysema. But when the abscesses are very numerous, there is something special in the appearance of the lung which requires to be accurately described.

Lobular pneumonia then becomes aggregated or pseudo-lobar: in other words, the inflamed lobules unite in large masses, invading, it may be nearly or quite, the whole of a lobe, as in the pneumonia of adults.

Two young children were attacked with acute pneumonia. The elder was taken to the Hôpital des Enfans Malades, where he died after a residence of a few days: the other, suckled by his mother, was taken to the Hôpital Necker, where he was placed under my care in St. Julia's ward.

The existence of pneumonia was unquestionable; but it seemed to be limited to the left side. There was heard on that side a very decided blowing sound, and also a considerable degree of reverberation of the cry. There was a rather coarse subcrepitant râle, and a little obscurity of the breath-sound. These signs continued to the last. On the right side, the respiration was feeble; and two days before death, we began to hear some subcrepitant râles unmingled with any blowing. There continued, however, ardent fever, and great oppression of the breathing.

At the autopsy, when the lungs were placed upon the anatomical table, a multitude of yellowish white spots were seen shining through the pleura, forming a striking contrast to the red colour of the hepatised parenchyma, which seemed to be stuffed with tubercles, some in a crude state and others in a state of softening. On making

a clean cut through a large mass of lung, a similar aspect was presented, with this difference, that the appearance of the parts was to a slight extent modified by the gushing of pus from the incised surfaces. By letting a small stream of water fall on the tissues thus altered, the water carried away some of the pus, and so disclosed to view an irregular cavity with imperfectly defined edges. As the little stream of water did not wash away the whole of the purulent matter, there remained a cavity not so well defined as before, and a very soft mass adherent to the parenchyma. Finally, among the portions of lung which presented at a first glance this general appearance of tubercles, there were some portions from which, although very friable, the water detached nothing. All around this, the parenchyma was hepatised.

A very little attention was sufficient to dispel the idea of the existence of tubercles. We had evidently to do with lobular pneumonia in four degrees: there was red hepatisation, affecting the great mass of the lobules; light-coloured hepatisation corresponding to the third degree of pneumonia in the adult; partial softening of the lobules which had passed to light-coloured hepatisation; and finally, complete softening of these same lobules—true peripneumonic vomicæ.

It was a somewhat remarkable fact, that these four degrees were observed in the left lung, which was the first as well as the most violently attacked: while the right lung, not attacked till two or three days before death, presented only the first two degrees.

During the autopsy, I took care to point out how much these lesions differed from tubercles; and to remark that one could not fail to recognise in the inflamed lobules the identical forms coexisting sometimes in entire lobes of the lungs of an adult.

Besides, the exceedingly acute character of the disease would indicate that it was a pure pneumonia; and although it be quite true that sometimes I have seen acute attacks of pneumonia prove fatal, in a few days, in children who had hardly ever previously coughed, and have, at the autopsy, found the lungs full of tubercles in different stages, it is not less true, that pathological anatomy furnishes means of distinguishing cases of pneumonia complicated with tubercles from cases in which the pulmonary tissue is studded with abscesses. Quite recently, I showed to all of you in this amphitheatre, the lungs of an infant at the breast, in which were thousands of little pouches filled with perfectly homogeneous pus. The infant had only been ill for a fortnight.

I now return to the consideration of what takes place in the adult. In adults, as I have already said, non-tuberculous, non-metastatic, purely inflammatory vomicæ are exceedingly rare: indeed, they are so rare, that during my first twenty-five years as an hospital physician, I never met with a case of this description. By one of those strange coincidences, however, which sometimes occur in practice, the two cases which we have seen together presented themselves to my notice during one week; and one of the cases has left some doubts in my mind. This lesion is so rare, that Laennec—whose authority is of great weight in such a question—affirms that in opening the bodies of several hundred persons who had died of peripneumonia during a period of twenty years, he had only five times met with abscesses in inflamed lungs. "Moreover," adds the immortal author of the *Traité de l'Auscultation Médiate*, "they (the abscesses) were inconsiderable, few in number, and scattered throughout the lungs, which presented the third degree of inflammation." Once only did he meet with a large abscess, such as we found in the first of our autopsies. Besides his own cases, Laennec says that he only knew of two other well-authenticated cases of abscess of the lung, notwithstanding the zeal with which pathological anatomy had been cultivated in France at the time he made that statement: one of the cases was communicated to the Academy of Medicine in 1823 by Dr. Honoré, and the other was published by Dr. Andral.[1] In support of this weighty testimony, I would adduce the statement of Professor Chomel, who, during twenty-five years, only twice found in the pulmonary parenchyma, purulent collections which did not seem to depend upon tearing by the pressure of the fingers at the moment of removal from the thorax[2]—an occurrence which frequently takes place in a lung infiltrated with pus.

A purely peripneumonic vomica is consequently an exceedingly rare affection in adults, and under such conditions as were present in the cases which have occurred in our wards. Bear clearly in your minds then these cases, as perhaps ere long you may find others like them.

Let me now, in a few words, sum up the history of our patients—a history interesting from many points of view. It is specially interesting in relation to the diagnosis of pneumonia; that is to say,

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[1] Andral:—Clinique Médicale; T. II, p. 313.

[2] Chomel:—Dictionnaire de Médecine, Paris, 1842. T. XXV, p. 151.

the real diagnosis of the disease, with which the clinic can alone make you acquainted, and which sometimes embarrasses the most experienced physicians—not that ordinary diagnosis, so simple and so easy, which may be learned theoretically from text books.

The first of our patients was a young man of robust constitution, twenty-six years of age. You first saw him lying in bed 19, and then in bed 7 of St. Agnes's ward. He had been ill four days when admitted to the hospital on the 24th March. His illness had commenced with a violent pain in the left shoulder, after exposure to a sudden transition from heat to cold, on leaving a ball. He, nevertheless, went to his work next morning; and although on the evening of that day, there was an increase of pain, and though with this, he also had fever, oppression of breathing, and cough, and although he had passed a sleepless night, he again returned to his usual employment on the 23rd March: he ate little at his midday meal; and in the evening, he had difficulty in regaining his lodging. During the night, the pain in the shoulder became still more severe, and with it there was also pain in the chest below the left breast: this increase of pain was accompanied by severe rigors. On the 25th, as I have already stated, he entered the Hôtel-Dieu; and I saw him next morning. He had intense fever; and his countenance indicated extreme anxiety. He was greatly excited: but the only complaint he made was of pain in the shoulder, which was increased by coughing and the exertion of breathing which was embarrassed and difficult. Although the movements of the shoulder-joint were painful, the pain in that region was not increased by pressure: he made but moderate complaint of the stitch in his side. There was no expectoration accompanying the cough. However, the intensity of the fever, and the very anxious appearance of the patient, made me think of a deep-seated pneumonia, inaccessible to our means of investigation; while on the other hand, the local pain suggested the commencement of an attack of articular rheumatism, which might perhaps declare itself next day. Following the latter indication, I caused ten scarifying cupping-classes to be applied to the seat of the pain. Since the evening, there had been diminution in the pain of the shoulder, but an increased severity of the stitch in the side, accompanied by extreme anxiety, by considerable embarrassment in the movements of respiration and coughing. Next day, these symptoms were very decided: the fever was more intense, and the excitement was greater. On percussion, only slight dulness was detected in the region of the

heart: no morbid phenomenon was revealed by auscultation. The pulmonary expansion was, it is true, interfered with, by the pain impeding the movements of the thorax. The sputa, which up to this time had been scanty and albuminous, now presented the yellow colour of barley sugar: they had a viscid consistence and were expectorated with difficulty. In the evening, the sputa had additional characteristics: they were sanguinolent, apoplectic, of a bright red colour, frothy, but still tenacious. My diagnosis of pneumonia was confirmed, although the physical signs were absent, except some dulness in the region of the heart detected by percussion: this dulness was limited to an extent of about ten centimeters from the nipple to the sternum, where a certain amount of arching was perceptible: pressure in this region occasioned acute pain. I came to the conclusion that there was pericarditis complicated with pneumonia. The autopsy, to the description of which I shall return, showed me my error in diagnosis: there was only extensive hypertrophy of the heart.

On the 23rd, I ordered twenty scarifying cupping-glasses to be applied to the region of the heart, and at the same time ordered a continuance of the precipitated sulphuret of antimony, which had been ordered on the previous evening, one gramme [15½ grains] being made up in ten pills. The expectoration, always difficult, had again changed its character: the sputa had the colour of the juice of prunes, was somewhat viscid, and adhered to the vessel. It was not till the 29th March, the fifth day of residence in hospital, and the ninth from the beginning of the illness, that we began to hear crepitant râles; but, when under the ear, they sounded so distant, so difficult to appreciate, that even their existence might be disputed. The general symptoms continued, and, moreover, increased in severity.

On the 30th March, the sputa had assumed a chocolate colour, without having any fœtor. On auscultating the chest, there was heard tubal blowing of very unequivocal character, although it seemed distant from the ear, and mixed with mucous râles of average coarseness. The resonance of the voice was broncho-egophonic. The dulness in the infra-spinous fossa was replaced, from the inferior angle of the scapula downwards, by exaggerated resonance, which could be elicited by strong percussion. This exaggerated resonance was so great anteriorly, down as far as the mamma, even when the percussion stroke was moderate, that the sound seemed to

be abdominal. I therefore said:—this man has pneumonia, a central pneumonia, which invading the anterior part of the lung has perforated the parenchyma, and determined an effusion of air and pus into the pleural cavity, establishing a communication between that cavity and the bronchial tubes. In a word, I diagnosed a peripneumonic vomica and hydrothorax.

On the 31st March, I observed that the vesicular murmur under the left clavicle had become weaker, and that towards the precordial region, there was heard distant amphoric blowing: lower down, the respiratory sound was absent. Behind, the vesicular murmur was so feeble, as to be scarcely heard at all in the scapular region: from the inferior angle of that bone, it gave place to very distant amphoric blowing: there was a somewhat obscure metallic resonance of the voice; and the sounds of the heart were heard posteriorly by conduction.

On the 1st of April, the expectoration, which in the evening had a chocolate colour, beginning to mingle with greenish sputa, became copious: it consisted of a pretty thick fluid, in which floated sputa which were frothy, not viscid, and free from any trace of blood. By auscultation, we could still hear amphoric blowing which came and went alternately, to which there was added a sound similar to that caused by bubbles of air traversing a fluid in a state of ebullition.

On the 3rd April, the severity of the general symptoms had so much increased, the state of matters had become so alarming, so desperate, that it was quite out of the question to think of making the patient change his position, to examine the chest behind. Expectoration was scanty; and in the spittoon, there were only four or five large, thick, greenish, purulent sputa. The pulse was 140, small, and intermittent. The skin, which was covered with a viscid sweat, presented a very characteristic cyanosed appearance. There was extreme anxiety, great oppression of the breathing, and an almost complete extinction of the voice.

On the 4th April, this young man was in a dying state. During the night and morning, he had brought up a great quantity of thick, creamy, greenish white, inodorous pus, which filled two spittoons. In the evening, low delirium set in; and on the morning of the 5th April, he expired.

At the autopsy, we found the lesions to which I have called your attention: we likewise found, as I have already stated, that the peri-

cardium was quite unaffected; but the heart, very bulky, occupying the space which I had marked out with the pleximeter, rested on the indurated lung, thus no doubt, giving rise to the thoracic arching and precordial dulness which had led me to suppose that there was pericarditis.

Our second patient was a man of thirty-three, and was also, like the former, of strong, vigorous constitution. For six months, he had been complaining of frequent headache and great lassitude On the 8th April, eight days before his admission to the Hôtel-Dieu, he felt more fatigued—more foundered [*plus fourbu*]—to use his own expression, than usual. He had been attacked with fever, unaccompanied by decided rigors, or stitch in the side. He said that at that time he had no oppression of the breathing, a statement however of no importance, as on admission he declared that he had no oppression, although I could see that it decidedly existed, respiration being short, quick, and anxious. He had a great deal of fever. On percussion, the sound was natural on the right side of the chest; on the left side, anteriorly, below the clavicle, the sound was exaggerated, skodaic. Behind, there was dulness from the top to the bottom of the chest. The vesicular murmur, normal on the right side, both before and behind, was likewise natural on the left side, where we detected the resonance; but behind, it was replaced by very intense tubal blowing and bronchophony: its maximum intensity was in the infraspinous fossa.

On the evening of the day upon which he came into hospital, he had only brought up one spit, which was saffron coloured, frothy, aërated, and non-adherent to the vessel in which he expectorated. On the morning of the 9th, his spittoon was filled one third with greenish diffluent sputa, some of which were brownish, reminding one of rusty sputa. I directed two palets [19 fd. ounces] of blood to be taken; and at the same time prescribed 50 centigrammes [nearly 8 grains] of precipitated sulphuret of antimony in five pills. In the evening of the same day, the blood, which had flowed freely, presented a diffluent appearance: the non-retracted crassamentum was covered with a thin greenish buffy coat. The pulse was compressible and soft, as in the morning. It was impossible to venture upon the farther abstraction of blood.

On the 10th April, the spittoon was still filled one third with very diffluent aërated sputa, having the appearance of dirty gum with a slight look of prune juice. The pulse had the same frequency, and

the same other characters, as on the preceding evening. There was extreme oppression of breathing, which increased during the evening. The patient fell into a drowsy state: his expectoration assumed a chocolate colour: his pulse, which was very soft, beat 136 in the minute. He died on the 11th April at 4 o'clock in the morning.

On opening the body, there were found the lesions of suppurative pneumonia, and perhaps what was an incipient vomica, as I stated to you.

To these two cases of peripneumonic abscesses, I shall add a third, narrated by Dr. Graves in his Clinical Lectures—in the lecture on "Abscess of the Lung."

Early in the spring of 1841, Dr. Graves was asked by Dr. Brereton to see with him at Sandford a lad between fourteen and fifteen years of age, who, a fortnight previously, had suffered from the symptoms of pleuropneumonia, with acute pain in the side and very violent cough. He had had the characteristic expectoration, as well as sputa of the colour of prune juice. The general symptoms, as well as the local inflammatory symptoms, were very severe; and did not yield to the treatment, which was both judicious and active. About ten days after Dr. Graves's first visit, matters were proceeding from bad to worse, and at that time the pulse was nearly 140: there was very great dyspnœa, excitement, jactitation, insomnia, and a cough ceaseless by day and by night. The case seemed desperate, and, hour by hour, death was looked for. Almost the whole of the right lung was involved in the pneumonia; and there was great dulness on that side. It is noteworthy, that in the first stage of the disease, crepitant râles were heard throughout the whole of both lungs.

The distinguished clinical professor of Dublin was well aware that this was a case of pleural vomica; but nevertheless, he is careful to mention the important fact that at the beginning of the illness, the patient had crepitant râles throughout the whole of both lungs, which hardly left any room for doubting the existence of pneumonia. I wish here, however, to make a reservation. Graves says:—"Crepitant râles *were heard*:" I should have preferred that he himself had heard them.

When matters were in the gloomy state now described, the patient was one night affected with very great difficulty in breathing, anxiety, and pain in the side: he was supposed to be at the point of

death. All at once, after a sudden effort, he brought up a large quantity of purulent matter; and immediately afterwards felt comparatively well. Next night, a similar struggle occurred, and was followed by a similar result. In the morning, when Dr. Graves saw the young man, he found him better in several respects; but he still had extreme debility with a great deal of fever and dyspnœa. On examining the right side of the chest, Dr. Graves found that the anterior part, from the clavicle to the base, as far down as the diaphragm, yielded a sound very different from that which it had previously rendered on percussion: it was then dull—now it was clear. This side of the thorax was evidently dilated; and, by the stethoscope, metallic tinkling was heard whenever the patient coughed or spoke. This satisfied Dr. Graves that there was a very large cavity in the lung, communicating in one direction probably with the bronchial tubes, and in the other with the pleural cavity. He looked on the case as hopeless. Fifteen days from that date, or, possibly, a little later, the expectoration again became purulent, and this recurred: but each time, the quantity was less, and the state manifestly ameliorated. In six weeks from the occurrence of the first purulent expectoration, convalescence was far advanced; and ultimately, the young man became strong and in perfect health.

The two cases which we have observed together in the wards, the case of Graves (who reports others of the same kind), the cases observed by Laennec, by Honoré, and by Professors Andral and Chomel incontestably prove the possibility of a purely inflammatory peripneumonic vomica. But it is not enough, Gentlemen, to detect a vomica at the opening of the dead body: we must endeavour to diagnose it in the living patient. Let us inquire, therefore, whether there are any other signs by which recognition of this diseased state can be made during life.

The elements of this diagnosis are generally scanty. The signs indicated by Laennec, the coarse, bubbling, mucous râles, manifestly cavernous, audible in the situation of the abscess: marked pectoriloquy taking the place of the bronchophony which previously existed; the respiration and the cough, previously bronchial, becoming cavernous; the blowing *being in the ear*, when the abscess is near the surface of the lung, and *muffled*, when a part of the wall of the abscess is thin and soft, these signs have very rarely been ascertained to coexist. They are very far from being so easy to distinguish as Laennec alleges: the pectoriloquy, and particularly the blowing heard

in the ear belong equally to pleural and peripneumonic vomicæ: this is the conviction which is left on the mind by reading the chapter in Graves's clinical lecture upon abscess of the lung. Graves reports three or four cases, occurring in the practice of himself and Stokes, of pleural abscesses, which opened into the bronchial tubes. On considering, however, what took place in the subject of our first case, on considering that the vomica was detected during the life of the patient, one is obliged to admit that some characteristic signs do exist. But in my opinion, there are some signs of greater importance than those pointed out by Laennec.

There has been, let us suppose, an acute, a very acute attack of pneumonia: then, at a later period, the individual expectorates a large quantity of puriform matter mingled with blood, and, in consequence of this admixture, presenting a chocolate colour: sometimes, the expectoration is diffluent, at times resembling the sputa of pulmonary apoplexy, and at other times like the fluid of certain hepatic abscesses situated in the substance of muscles. It is a mixture of blood and pus. New stethoscopic phenomena become observable at the same time: in a limited portion of lung, there is amphoric respiration, a gurgling bubbling sound; and along with this there is sometimes a metallic tinkling which passes into the cavity.

It was not from the expectoration alone that I formed my diagnosis in the first case. The sputa, at first hæmorrhagic, became, however, of a chocolate colour, that is to say, mixed with pus and blood. On the sixth day from his admission to the hospital, which was the tenth day of his illness, symptoms of hydropneumothorax all at once showed themselves; then also, was observed profuse purulent expectoration; and then, too, it was that I diagnosed the vomica. To arrive at such a conclusion, however, it is necessary to have a conjunction of all these signs—a peculiar expectoration, amphoric blowing, and metallic tinkling.

In the case of our second patient, in whom I thought a vomica was beginning to form, in whom the pulmonary abscess was still filled with the putrilaginous matter which you saw at the autopsy, we only discovered the existence of pneumonia in its third stage: and you can understand how difficult the diagnosis was in other respects, the burrow not being as yet empty, and neither communicating with the bronchial passages, nor with the pleural cavity.

Therefore, it is, that the quantity of the sputa, the sudden increase in their quantity, their special character, their becoming diffluent after

having been viscid, are the circumstances which guide us to the diagnosis of an open vomica, whether the opening be simply into the bronchial tubes (as in our case and in Graves's case), or into the pleural cavity as well as into the bronchial passages. The time at which this communication is established is perhaps the capital element in the diagnosis. It is almost impossible for a peripneumonic vomica to remain long without opening. An abscess formed in the parenchyma of the lung will try, like every other purely inflammatory abscess, to open externally, and the pus will necessarily find an exit by the divided and ulcerated bronchial tubes, which correspond to the cavity of the abscess: if at the same time it opens into the pleural sac, the peripneumonic vomica does not with less rapidity find an outlet by the bronchial tubes. Indeed, there is not on record any case in which the formation of the opening has occurred later than the twentieth or twenty-fifth day. Abscesses which open on the fortieth, fiftieth, or sixtieth day, are abscesses of the great pleural cavity, or abscesses between the lobes of the lung. In fact, in numerous cases, we find between the lobes of the lung a collection of fluid sometimes serous, sometimes sero-purulent, which is imprisoned between the lobes by false membranes closing in the interlobular fissure: these collections, to a certain extent independent of the pleural cavity, nevertheless belong to the pleura: like purulent collections in the pleural cavity, they may find an outward passage through the bronchial tubes by perforating the pulmonary parenchyma; and when this state of matters exists, the patient presents all the symptoms of pleural vomicæ. But as there is necessarily an absence of the signs of effusion into the great cavity, as there is only a dulness which seems to depend upon the state of the lung, the conclusion arrived at will be that there is a peripneumonic vomica. These supposed abscesses of the lung, however, are, I repeat, very long in opening—six weeks, two months, three months, or sometimes, it may be, four months from the commencement of the pleuropneumonia. This original pleuropneumonia causes the mistake: it has been followed in all its phases, and that which has been ascertained to exist has appeared to be its sequel, and to be related to the pulmonary and not to the pleural lesion. You are, under such circumstances, the more inclined to believe that there is pulmonary vomica—the sounds of gurgling seeming to be limited to the lung, and not being accompanied by the usual signs of hydropneumothorax.

That which takes place in interlobar pneumonia, occurs also in cir-

cumscribed pleurisies of the great pleural cavity itself. From the numerous examples you have seen, you are aware that under certain circumstances adhesions take place between the pulmonary and costal pleura, that a pleurisy at the base of the lung terminates in resolution, while a pleurisy at the upper part of the lung does not enter upon resolution, but proceeds to suppuration. There then supervenes a lesion difficult of recognition. Suppose that a pleuropneumonia has previously existed:—the pulmonary inflammation was manifested by bloody mucous expectoration, and afterwards the sputa assumed a rusty colour, and looked like apricot marmalade: by the stethoscopic, pathognomonic crepitant râles were heard: the pleurisy itself had been characterised by the violent stitch in the side, differing from that sensation of weight and pang which the old authors connected more particularly with the existence of peripneumonia. Adhesions were formed between the inflamed lung and the costal pleura. The remaining pleuritic effusion between the adhesions first became seropurulent, and then entirely purulent. There can still be perceived in the situation corresponding to the dulness and the blowing, a very considerable bellows-sound notwithstanding the great quantity of effusion, for, as you are aware, a considerable amount of blowing is not inconsistent with great effusion. This then was a case of circumscribed pleurisy; and in that situation, the compressed lung, becoming squeezed on itself, was at last completely flattened by the effusion.

It then becomes very difficult to follow the evolution, which leads to the belief that pulmonary induration exists alone, in consequence of the stethoscopic signs being bronchial blowing, vocal resonance, and sometimes even coarse gurgling râles, phenomena which occur within the as yet unflattened bronchial passages, and are transmitted across the condensed pulmonary parenchyma and the effused fluid within the pleural cyst. Under such circumstances, the diagnosis is —pneumonia, which has become chronic. However, in two or three months from the beginning of the attack, the patient brings up a large quantity of pus by the mouth—in the literal meaning of the term, he has a vomica—he vomits: you then hear within the chest, gurgling and the bursting of large bubbles, as well as metallic tinkling; and you come to the conclusion that in the indurated portion of lung, a cavity has been formed, that cavity being constituted by the pleura. In this case, the sole element of differential diagnosis was the time of the opening of the abscess—the time of appearance of the vomica; as I have just been saying, of all the signs which

have been mentioned as diagnostic of pulmonary abscess, time of appearance is certainly the most important.

By paying special attention to this sign, the mistaking pleural for pulmonary, and pulmonary for pleural abscesses may be avoided, particularly if the patient has been under observation from the beginning of his attack. On the other hand, when the patient is not seen till an advanced period of his malady, such mistakes though much more readily committed, may still be avoided. Indeed, generally speaking, pleural effusion is easily recognised: complete dulness, and distension of the chest, never—absolutely never—accompany pneumonia; and there is nearly always absence of thoracic vibration: these are phenomena sufficiently characteristic. It is true that in some exceptional cases, thoracic vibration is absent in pneumonia; and it is also true, that thoracic vibration may exist in some cases of pleurisy, as, for example, in pleurisy accompanied by bronchophony. But when, in addition to the phenomena now pointed out, we meet with others, such as the crushing up of the mediastinum, and the pushing over of the heart to the unaffected side, the pressing down of the liver or spleen, one can have no doubt as to the existence of extensive pleural effusion, nor will there be any chance of mistaking it for pneumonia. But if, in these cases, the patient has suddenly vomited a large quantity of pus, you may, without any farther examination of the chest, without using stethoscope or pleximeter, affirm that the pus comes from the pleura. Auscultation will generally confirm this diagnosis, by enabling you to recognise the signs of hydropneumothorax.

This is a point upon which I have insisted, when discussing the history of pleurisy and pneumothorax. At present, I shall only recall to your recollection the fact that these large pleural purulent collections may open into the bronchial passages without necessarily causing any great harm to the individual.

Three years ago, Dr. Bordes called me to see with him in consultation a fruiterer of the Rue des Gravilliers: our appointment was, to meet at this man's house at half-past ten in the morning. Dr. Bordes had detected considerable effusion in the chest, dating back two months and more: he begged me to bring with me the instruments necessary for performing paracentesis of the chest; and consequently I went prepared to operate. On my arrival, the patient showed me a salad dish containing five litres of pus, which he had vomited during the night. During the day, he continued to bring up pus in

large quantities, and within less than a week, he brought up eleven litres by exact measure. He continued for three weeks or a month to *vomit* pus, to use his own expression. At present, he is in excellent health.

Large pleural vomicæ, then, may, like pulmonary vomicæ, open into the bronchial passages; but, irrespective of the signs which I have given you, the very quantity of the pus brought up will not allow the practitioner for one moment to have any doubt as to the nature of the case. No abscess of the lung can contain a litre of pus: that I hold to be impossible, but a pleural abscess may contain two, three, or four litres; moreover, as the pus is renewed day by day, the quantity brought up by an individual may be much greater. For example, Legroux mentions the case of an individual, who—during a period, it is true, of considerable duration—brought up from 42 to 43 litres, actually measured; and at a meeting of the Medical Society of the Parisian Hospitals, in 1854, I read an account of the case of one of my patients, a girl six years old, operated on for empyema, who, within a period of rather more than six months, brought up pus estimated at 200 grammes a day, making the enormous total weight of 40 kilogrammes.

The capital difference between the quantity of pus expectorated in cases of pleural and pulmonary vomicæ simplifies their differential diagnosis. The difference in the quantity of pus expectorated, and the different period at which the vomica opens, constitute the essential elements of the diagnosis. In children, however, the last-mentioned diagnostic element may be wanting.

In children, purulent collections in the pleura may open very quickly into the bronchial tubes. Suppose a case of pleurisy in which the diagnosis has been made with exactitude at the beginning of the attack. Effusion has been detected, and its increase observed: symptoms soon show themselves indicating that the effusion has become purulent: at last, about the fifteenth, twentieth, or eighteenth day of the attack, the patient brings up pus in large quantities. The existence of a pleural vomica is in such a case established beyond the possibility of mistake. In the adult, cases of this description are of exceptional occurrence; but they are sometimes met with in persons of suppurative diathesis. In puerperal women for example, you may have rapid formation of pleural abscesses; and you may also have them opening into the bronchial tubes very rapidly, much more rapidly than in ordinary cases. Under such circumstances, there

might be great difficulty in establishing the diagnosis, owing to the element of doubt introduced by the expectoration of pus ; but if we have seen the cases when the first symptoms set in, if we have then perceived the presence of a suppurative pleurisy, the connection of the suppurative phenomena with the puerperal condition and general symptoms of the patient, will suggest themselves to your minds ; and when the vomica bursts externally, you will realise the necessity of being guarded in your prognosis.

To complete my remarks on pulmonary abscesses, let me add a few words on their pathological anatomy and mode of termination. In respect of their pathological anatomy, I would call your attention to the characters which distinguish purulent collections originating in acute simple inflammation from the vomicæ met with in phthisical subjects. On this point, I cannot do better than quote the exact words of Laennec. He says :—

"Although in some cases, the colour and aspect of tuberculous matter are very similar to those of pus, tuberculous matter generally differs from pus by containing an admixture of fragments of softened friable tubercle. Moreover, the circumscribed character of the cavities formed by the softening of tubercle, the firm consistence of their walls, the soft false membrane by which they are always lined, and the semi-cartilaginous membrane which sometimes succeeds to this false membrane, are sufficient to characterise a lesion very different from the above-described abscesses."—I would add, Gentlemen, to this description of Laennec, that one never meets with a tuberculous vomica, without finding at the same time numerous tubercular masses at different stages in one or both lungs.

Regarding the prognosis, I cannot speak from my own experience alone; because, as I have already told you, I never had a case of pulmonary abscess till I met with the two which have given occasion to the present lecture. Judging from these cases, and from what has been written on the subject, I believe that pneumonia terminating in pulmonary abscess is generally mortal. No doubt, Laennec, Graves, and others show the possibility of recovery by the abscesses opening into the bronchial tubes, and cicatrisation of the cavities taking place ; but without denying the possibility of cure in this way, I agree in opinion with those physicians who regard such cases as altogether exceptional.

In the treatment of cases of pulmonary abscess, as you can under-

stand, Gentlemen, there is nothing special to be done. Up to the time that the existence of the pulmonary abscess is ascertained, the treatment in no respect differs from that of ordinary pneumonia: and when we ascertain that the abscess is actually formed, intervention can avail nothing, as the abscess is situated beyond the reach of our remedial resources.

LECTURE XXXVI.

TREATMENT OF PNEUMONIA.

Simple Pneumonia without any Complication.—Expectant Medicine.—Local and General Bleeding.—Blisters.—Antimonial Preparations, particularly the Precipitated Sulphuret [Kermès] in large doses according to Rasori's Method.

Gentlemen:—I am certain that in all the different hospitals which you frequent, there are no wards in which the local and general abstraction of blood is resorted to so cautiously [*aussi sobrement*] as in mine. This arises from the fact that as yet the necessity, the utility even, of bleeding does not appear to me to have been made out so clearly as is believed by the majority of physicians, to whom the denial of the efficacy of abstracting blood in pneumonia would seem the denial of a demonstration.

Even in pneumonia, a disease which according to received ideas, demands bleeding more than any other, you seldom hear me order it. If I sometimes have recourse to it, it is because it seems to be indicated by certain complications of the case, rather than with any view to combat the ordinary inflammatory element of pneumonia: the cases in which I resort to it are too few in number, to weaken in any degree the general applicability of the rule by which I have been guided in this matter for many years.

Gentlemen, this practice differs so essentially from that which I may say is almost universally followed—from that which is followed by the majority of your teachers, my colleagues as hospital physicians, and by the classical authors whose works are in your hands—from that which is accepted as orthodox by non-medical public opinion, which does not recognise the possibility of inflammation of the lungs being cured without the abstraction of blood—that I am bound to explain myself to you fully on this subject, and to expound my views on the treatment of pneumonia.

But before grappling with the merits of this interesting question, it is essential clearly to define the terms employed in the discussion.

Pneumonia is not uniform in its character: the forms which it assumes, its greater or less intensity and extent—the influence of the prevailing medical constitution—the personal specialities of the patients in respect of age, sex, temperament, and previous health—the diseases which may complicate pulmonary inflammation, and the unfavourable conditions which may supervene during its course—all demand particular inquiry on the part of the physician. He must take special account of all of them, for they greatly modify the discase, and are also the source of much diversity in the therapeutic indications.

For the present, I shall delay consideration of that particular form of pneumonia, which I very willingly call *catarrhal pneumonia*, and which is observed in the early years of infancy and childhood, which is in the adult one of the most formidable epiphenomena of serious fevers; and also, particularly, of measles and whooping-cough, as, on former occasions, I have pointed out to you.

I shall also, for the present, say nothing of pneumonia complicated with symptoms giving it a special stamp: it will be sufficient for me to name *bilious pneumonia*, so remarkably described by Stoll, but which is seldom met with in the present day, a circumstance probably explained by the existing medical constitutions differing from those under which Stoll observed; *ataxic* and *adynamic pneumonia*, which take their names from the predominance of nervous symptoms; *arthritic* and *rheumatic* pneumonia, unquestionably species of pneumonia, though their existence has been denied by some.

The kind of pneumonia of which I wish to speak to you to-day is *peripneumonia vera*—simple legitimate pneumonia—that form of the disease which most frequently presents itself to our notice, and generally supervenes from an accidental cause, generally from a chill.

I shall now rapidly sketch its principal features. The period of incubation is either short, or has had no existence. The malady is generally ushered in by a shivering fit; but this phenomenon is sometimes absent. The local phenomena generally open the scene. There is a stitch in a part of the side of variable extent: it is complained of by the majority of patients as existing at the base of the lung, more particularly under the nipple; it is generally increased by the inspiratory movements and by coughing; and is intensified by pressure. The respiratory movements become accelerated; and there is

oppression of the breathing—much more, however, in appearance than in reality. The cough, at first dry and distressing, is almost never absent. The local phenomena are accompanied by intense fever: the skin is hot, having sometimes a burning dryness, but being more usually covered with a greater or less amount of perspiration. The patient complains of a feeling of discomfort, general bruising, and headache: the countenance appears flushed and excited: the tongue is covered with a white saburral coating, and is sometimes yellowish towards the base: there is intense thirst, and no appetite for food. Bilious vomiting is often the first symptom: diarrhœa is very common at the beginning of the illness; generally, numerous herpetic bullæ appear on the lips and around the nostrils.

During the first twenty-four hours, the cough is generally dry, as I have just stated, or at least the expectoration which accompanies it has as yet nothing characteristic of the malady; but on the following day, it begins to assume more and more that appearance which is destined to become specific. *Peripneumonic sputa* are viscous, glutinous, semitransparent, and minutely acrated: although, as yet, they have not much of the rusty character, nor are they, as a rule, very sanguinolent, some of them at least present occasional striæ, or a small compact nucleus, the colour of which passes from amber-yellow to the tint of barley-sugar. This coloration, due to the admixture of blood with the mucous secretion, becomes more and more marked, presenting different shades, particularly that of apricot marmalade, of saffron, and of iron rust. At the same time that the sputa undergo these changes, they become more abundant, coalesce into one mass in the spittoon, and form a sheet, semitransparent like the cornea. The sputa strongly adhere to the bottom of the vessel. They are of themselves sufficient to enable us to recognise the nature of the disease; but other physical signs, the existence of which we are able to discover by auscultation and percussion, are pathognomonic of inflammation of the pulmonary parenchyma.

These signs do not show themselves during the first twenty-four hours: at least, percussion furnishes no positive element of diagnosis, thoracic resonance not being sensibly modified, and auscultation affording little more than negative results in respect of changes in the respiratory sounds. But on the second day, a dulness more or less appreciable reveals itself in the parts originally affected; and there are also heard in that situation anomalous sounds which become more and more decided.

The earliest of these sounds is a fine and very equal crepitant râle, which is heard during inspiration: the shocks imparted by the cough, so far from causing it to disappear, make it reach the ear of the auscultator in blasts. This crepitation tells us that there is engorgement of the lung; and I need not here discuss the theories which have been advanced to explain the production of this crepitation.[1] This râle is accompanied by, and soon afterwards replaced by *bronchial respiration*, which has also received the name of *bronchial blowing*. This blowing sound presents different varieties: sometimes, it is distant from the ear; and at other times, on the contrary, it is harsh and noisy; sometimes, it is large and diffuse, and at other times, limited and resounding, constituting tubal blowing. It generally begins by replacing the sound of expiration only, but it afterwards invades that of inspiration, and then accompanies both expiration and inspiration. The voice, which has hitherto been only slightly resonant, now loudly resounds in the bronchi, and is transmitted to the ear by the hepatised pulmonary tissue, which being more dense than the remainder of the lung, becomes an excellent conductor of sound. This *bronchophony* is never more marked than when it occupies the root or summit of the lung, where the bronchial tubes are larger than elsewhere.

Crepitant râles are also often heard within the same space in which the blowing sound and vocal resonance are audible.

These characteristic physical signs of the disease which we are now studying, sometimes escape detection, from the inflammation being confined to a central situation in the lung, or to a sufficiently careful examination not having been made. You may be able to hear the morbid sounds only in the axilla.

The general symptoms continue along with the local phenomena;

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[1] Without desiring to diminish in the smallest degree the glory of Laennec, to whom belongs the whole of the honour of having both discovered auscultation, and of having at one stroke as it were brought it to a very high pitch of perfection, it will, I think, be interesting to quote in this place the following passage from Van Swieten, according to whom it would appear, not only that the ancients had an idea of the existence of the crepitant râle, but likewise gave a theoretical explanation of its cause. Van Swieten says:—

"Plerumque tunc simul adest ingratus inpectore strepitus, qui fit vel ab acri muco hic collecto, irretitio: vel a vesiculis pulmonum siccis, hincque *crepitantibus* instar corii rarefacti, dum inspirando extenduntur."—G. Van Swieten, *Comment. in Herm. Boerhavii Aphor.*, § 826, *Peripneumonia Vera*, *T. II*, *p.* 659.

and they never show themselves in a more decided manner than from the fifth to the eighth day. The fever is more intense at that period. The flushing of the face is at its height, and is greatest over the cheek bones. The redness in this situation was looked on by our predecessors as one of the characteristic symptoms of pneumonia.

Physicians, encouraged by the alleged success of the Hahnemann sect in treating pneumonia, and following also other examples, have submitted their pneumonic patients to the expectant system. This method was adopted long ago by Magendie; and there are doubtless some among you who have heard of the recently published works of MM. Dietl of Vienna, Niemeyer of Griefswald, Schmidt, &c., and of the cases reported by Dr. Laboulbène: many must have read the posthumous treatise of Legendre, intituled—"*De l'Expectation dans la Pneumonie Franche.*"[1] Well, then, Gentlemen, these experiments have made us acquainted with the natural course run by purely inflammatory pneumonia in a great number of cases. Generally speaking, there is a tendency to spontaneous recovery, which usually occurs between the ninth and eleventh days.

According to Dr. Bourgeois of Etampes,[2] who, for twenty-five years, has abstained from all active therapeutic treatment in pneumonia, (and has published a short paper on the subject), there is, at the eighth day, a marked tendency to diminution in all the symptoms, in the cases which do well. At that period, the sputa are less coloured and less viscid: respiration is a little less embarrassed: there is no longer pain in the side: there is a diminution in the thickness of the saburral coating of the tongue: there is a return of sleep, which, during the previous days, had been absent or had been replaced by a state of constant drowsiness: towards the close of the day, the drowsiness ceases, and the patient begins to feel the want of restorative measures.

On the ninth day, there is almost always improvement: though there is more cough, it is looser: the sputa, albuminous rather than gelatinous, are nearly always colourless: the stitch in the side has quite ceased, unless it be that it returns during severe coughing fits, or on taking a deep breath: the tongue has become clean: there is a decided appetite for food: the urine, which was of a scalding character and scanty, during the acute stage of the pneumonia,

[1] Legendre:—*Archives Générales de Médecine*, for September, 1859.

[2] Bourgeois (d'Etampes):—*Union Médicale*, for 3rd January, 1860.

becomes abundant, and nearly normal in character, having no deposit and being devoid of turbidity, appearances which rarely show themselves except during convalescence: in a word, the symptoms of the disease disappear, *while its physical signs remain in their plenitude.*

On the tenth day, the patient enters upon complete convalescence. At the end of the second week, should nothing occur to impede the progress of recovery, the patient is in a state to resume his ordinary occupations, provided they are not of a fatiguing nature. Nevertheless, upon auscultation at this period, we still find the dulness, and also the crepitant râle which had replaced the tubal blowing, but it is the crepitant râle or, to speak more correctly, the subcrepitant moist "*râle de retour,*" as it is called, indicative of the return of the air into the pulmonary vesicles, whence it had been expelled by hepatisation. Several weeks will often be required for the complete disappearance of the signs of engorgement of the lung.

In simple pneumonia the temperature rises rapidly after the shivering fit which occurred at the beginning of the attack: it often reaches, and sometimes goes above 39° during the early days of the disease. It then continues at that point, with however slight irregular oscillations. The case is serious when the temperature rises to, or ascends above, 40 degrees. The fever generally abates on the 5th, 7th, or 9th day: this abatement takes place abruptly, and with rapidity: in twelve hours, or in thirty-six hours at the most, the temperature falls three degrees or more. As soon as the normal temperature is attained, resolution begins, and the patient may be looked upon as convalescent. At defervesence, it occasionally happens that the temperature falls for a very short time below the natural standard: in the cases in which this occurs, there is a temporary collapse.

These data, derived from numerous researches of Wunderlich, you have had an opportunity of verifying in a patient who occupied bed 20 of St. Agnes's ward. This man, aged 27, addicted to drinking and affected with alcoholic tremors, had extensive pneumonia at the upper part of the right lung. From the first days of his attack, the urine was found to contain a considerable quantity of albumen: he was delirious on the night of the sixth day of the disease, and during the seventh and eighth days, the delirium continued. On the ninth day, however, a complete convalescence set in, and by the

eleventh day, there was no longer any albumen in the urine. Here is what occurred in respect of temperature. On the fourth day, the day on which the patient was admitted to the hospital, his temperature was 39°: on the morning of the fifth day, it had risen to 40°.2, and in the evening of that day it fell to 39°.6, under the influence of antiphlogistic treatment: on the sixth day, during the whole day, morning and evening, it kept at 40°.4: on the evening of the eighth day, it fell to 39°.8, then on the morning of the tenth day, it fell to *thirty-six degrees and six tenths*—which is below the normal standard, to rise again to 37° and 37°.2, where it definitively remained.

I am unwilling to quit this interesting subject of temperature, without pointing out to you an important clinical peculiarity. At the beginning of an inflammatory affection of the chest, when there is stitch in the side, it is occasionally very difficult by unaided physical signs and the reaction to ascertain whether the inflammation is pneumonia or pleurisy: well, if from the first days of the attack, the pulse rises rapidly, reaching 39°, or, *a fortiori*, if it rise to a higher figure, pleurisy may be excluded, and pneumonia diagnosed: and likewise, the continuance of a relatively low temperature makes it probable that there is pleurisy, or, at all events, it excludes the idea of the existence of simple pneumonia.

Are we obliged to conclude, that the treatment of pneumonia ought to be expectant, because recovery takes place spontaneously in a certain number of cases? I think not: and, moreover, when I find myself confronted with this disease, I cannot remain an inactive spectator. Whenever I am called in to a patient suffering from pure and absolutely uncomplicated pneumonia, I lose no time in intervening by *antiphlogistic treatment*.

As I stated at the beginning of this lecture, I very rarely have recourse to the *abstraction of blood*, local or general. When there are symptoms of great general plethora, threatening to complicate the progress of the disease, I sometimes, though very rarely, cause a vein to be opened. After a single bleeding, however, of from four hundred to five hundred grammes, I seldom require to repeat the proceeding. To remove or moderate the stitch in the side, when the pain is excessive, I prescribe cupping glasses to be applied to the seat of pain, abstracting or not abstracting blood as the case may be; or I inject some drops of a solution of atropine into the subcutaneous cellular tissue: but that is the limit within which I bleed in

pneumonia. Bleeding, once extolled by physicians of the highest repute, employed formerly in one form or another by almost all practitioners, is energetically objected to in the present day. Some clinical physicians not only deny its efficacy, but even regard it as generally injurious. The only cases in which they do not regard it as objectionable are those in which the inflammatory symptoms are accompanied by excessive reaction, such as intense headache, somnolence, and great dyspnœa. In such circumstances, even, though they sanction bleeding as a means of affording temporary relief, they insist on the necessity of drawing blood in moderation. Though in these cases, they admit that bleeding may prove palliative, they deny that it is ever a means of cure, far less do they admit that it ever has the power which has been assigned to it of cutting short the disease. And again, the physicians to whom I refer, looking to the statistics which have been drawn up to elucidate this question, have come to the conclusion that there has been a greater mortality in pneumonia among those who have been bled, than among those who have not been bled: and consequently, they say that bleeding has been the cause of the numerous deaths from pneumonia, notwithstanding the immediate temporary benefit which it affords.

You have heard Dr. Beau,[1] in his clinical lectures, develope these ideas regarding the unfavourable effects which bleeding produces upon pneumonic patients. In citing to you cases from his own practice, in supporting his views by other cases quoted from the works of numerous French and foreign physicians, my honorable colleague of the Hôpital de la Charité has endeavoured to explain them to you by entering into physiologico-pathological considerations.

Although I also call in question the advantages of the abstraction of blood, the usefulness of which, particularly in the treatment of pneumonia, seems to me to have been vaunted beyond measure, I cannot agree with those who are the detractors of bleeding. Although I do not admit its utility in the majority of cases, allowance being made for the peculiar medical constitutions through which we have passed in recent years, yet, at the same time, I deny that it brings in its train the disastrous consequences ascribed to it, provided it be practised with due moderation. If, as a general rule,

[1] Beau:—Gazette des Hôpitaux, for the 6th and 8th September, 1859.

I abstain from prescribing bleeding, it is not because I believe it to be the cause of the frequent deaths which have been attributed to it, but because my experience has taught me that it seldom shortens the duration of the disease, and frequently retards complete return to health, by weakening the patients, and prolonging the period of their convalescence.

Antimonials have not these drawbacks. Their antiphlogistic properties are as unquestionable as those of bleeding: the only difference is that they act in a different manner. While sanguineous evacuations suppress inflammatory action by removing the materials which constitute its aliment, while they exhaust the disease by exhausting the patient, antimonial preparations act in a wholly different manner, and never bring along with them the extreme prostration which often accompanies the convalescence from pneumonia treated by repeated bleedings.

This action of antimonials has been explained in many different ways. Rasori explained it by saying that these medicines *exhausted the diathesis of the stimulus;* but he did not define very well the meaning which he attached to that expression. According to Dance and Chomel, antimony does not possess any specific property. When there is a complete tolerance for it, they say that it is inert; and when it acts as an emetic or purgative, they hold that its action differs in no respect from that of any other evacuant. This opinion comes very near to that of Broussais, the eminent professor of the Val-de-Grâce, who says that antimonials ought to be regarded as revulsives even more powerful than the blisters and the sinapisms, which are applied to the skin, inasmuch as they act upon a larger surface, and moreover often excite profuse discharge from the gastro-intestinal mucous membrane.

Gentlemen, this is not the place to repeat a discussion which you will find given very fully in the article on *antimony* in my treatise on therapeutics. I nevertheless ask you to allow me to add what is there said on this head. As a general rule, I attach very little importance to explanations of the therapeutic action of medicines. In therapeutics, I only see two things—the administration of the medicine, and the result of that administration. As for the intermediate phenomena, they escape our observation, and perhaps will always continue to do so. Notwithstanding, I have hazarded my theory as to the mode in which antimonial preparations act—holding it, however, as very cheap, and being quite willing to abandon it for

any other which may appear to me to be more in conformity with facts. I asked myself whether we might not grant that antimony exercised a special toxic action on the heart and respiratory organs, either directly or through the medium of the nervous centres, just as many medicinal substances have unquestionably a special action on certain organs. The existence of this specific action of antimony appeared to me to be demonstrated by its physiological effects as manifested by the pulse becoming slower and weaker, and by the breathing becoming slower. This fact being established, the therapeutic effects of antimonials in pneumonia may be ascribed to a diminution in the quantity of blood sent to the inflamed lungs, which, by having a less degree of activity, are in a state analogous to that in which the surgeon places a fractured limb—that is to say, in a state of relative if not in a state of absolute repose.

Experiments on animals confirmed the views which I had formed as to the toxic action of antimonials upon the heart and organs of respiration. I had long previously enunciated the opinion I have now stated, when—in 1856—the experiments of Ackerman, and afterwards those of Pécholier demonstrated its correctness. If, as has been done quite recently in confirmatory vivisections, by my learned colleague Professor Sée—if a solution of tartar emetic be injected into the veins of rabbits or of guinea-pigs, there are soon observed a decided lowering of the pulse, diminished arterial pressure, and frequently, likewise, irregularity in the pulsations. Along with this condition of the pulse, there is a great depression of the vital powers. The lowering of the pulse and the diminution of arterial pressure are due to diminished frequency and diminished energy in the contractions of the heart: and this may be attributed to a direct, and to a certain extent paralysing, action of the tartar emetic upon the cardiac ganglia, which are the automotor ganglia of the organ.

Whatever may be the explanation of their action, the utility of antimonials in the treatment of pneumonia is now generally admitted. Violently and unfairly attacked by many, and inordinately extolled by others, the tartar emetic treatment has at last taken its place in the domain of theraputics. But if even persons who were the most incredulous have become convinced of the efficacy in pneumonia of tartar emetic in large doses, it is not so with some other preparations of antimony. The kermès, which you see me prefer to tartar emetic (for reasons which I am about to state), and the white oxide

of antimony, which some believe to be inert, have not as yet acquired the same rights of citizenship.

Nevertheless, there is evidence to show, that in the treatment of peripneumonia, the kermès is in no degree inferior to tartar emetic. It has, moreover, this advantage over tartar emetic, that it is much less irritating, and much more rarely produces the inflammatory affections of the throat and gastro-intestinal canal which prevent the continuance of the tartar emetic for a period sufficiently long, to bring about resolution of the pulmonary inflammation, and particularly to prevent its recurrence.

In respect of the *white oxide of antimony*, numerous cases have demonstrated to me its beneficial influence, particularly in the treatmemt of the pneumonia of children. There is, however, a necessity for giving it in large doses: and the actual good results may unquestionably be obtained by smaller doses of the kermès.

Some persons have seemed surprised—their surprise, however, being more seeming than real—that I have appeared to abandon the use of certain antimonial preparations which I lauded at a previous period of my professional career: narrow-minded and ill-natured individuals have made this circumstance the ground of bitter and insulting criticism. They might have spared me, had they remembered a great law of therapeutics, to the effect, that *the medical constitution has an immense influence on the action of remedies.*

This grave question approaches too near the domain of clinical medicine not to be here discussed, just as in former times I discussed it before my classes at the Faculté de Médecine, and as Dr. Pidoux and I have discussed it, in our *Traité de Thérapeutique.*

Medicinal substances, when administered to human beings, may be correctly regarded as morbific agents similar to those which commonly beset us. Have the ordinary morbific agents always the same mode of action? To experience we must refer this question for a reply.

A man, during a particular epidemic constitution, is exposed to the inclemency of the atmosphere: he takes pneumonia, and at a later period, articular rheumatism, pleurisy, or colitis. Here, therefore, there is the same cause determining an inflammation to different organs. This happens so frequently, that it is impossible for any one to deny that it is a common occurrence. During the cholera epidemic of 1832, causes apparently the least calculated to

disturb the digestive functions, produced diarrhœa, and sometimes a sudden invasion of cholera. Two years later, during the influenza epidemic, the same cause which had before produced cholera gave rise to a special form of catarrh. No change had taken place in the cause: it was identically the same. Why, then, did it not produce the same effects?

In considering the way in which a cause acts, two things of equal importance have to be borne in mind: first, there is the nature of the cause, which is always the same: and second, there is the support of the cause, that is to say, the economy within which the cause operates, which is subject to infinite variety, and reacts in virtue of idiosyncrasy, and also in virtue of an accidental tendency which exercises an immense influence. It is this accidental tendency [*disposition accidentelle*] which, when distributed at the same time to a great number of persons in the same district is called the *epidemic constitution*: it bears the same relation to the general population that *idiosyncrasy*, or *special constitution*, bears to the individual.

When, therefore, the whole, or nearly the whole of a population have one *common accidental constitution*, called *medical* or *epidemic constitution*, the same cause which but for this constitution would have produced certain known effects, will produce very different effects, because the support of the cause—the economy—has a different bias, in virtue of which its reaction is different.

The medicine administered to the sick man not only finds him suffering from the particular ailment for which it is prescribed, but also, finds him under the influence of the common or epidemic constitution, which necessarily modifies the effects of the ailment. Suppose, for example, that a choleraic constitution prevails in a district. If mercurial frictions are employed within that district, in puerperal fever, or articular rheumatism, exceedingly serious gastro-intestinal symptoms may supervene; and the mercury, diverted from its normal action, irritates the intestine before manifesting its ordinary effects.

The aptitude of the illustration now given is palpably evident: but the influence of the medical constitution is not less constant in a host of other circumstances in which the manifestations are less clear. Testimony to that effect can easily be collected from the writings of all intelligent medical observers of the times anterior to our own.

In the present day, a physician makes himself the champion of a therapeutic idea, or rather of an experimental idea, which is not the same thing. He goes on for many years submitting all his patients, irrespective of age, sex, temperament, and medical constitution to identically the same treatment: and month by month, and year by year, gravely registering the numbers of the deaths and recoveries, he finally deduces from these statistics therapeutic laws which he looks upon as irrefragable. It matters little to him that one year he had a frightful mortality to deplore, and that in another he had to congratulate himself upon a great number of recoveries. To him it is simply a question of figures: he adds up, and calls the result—a law!

If you ask him how it is that fifteen years ago, he lost one out of three patients, while now he only loses one out of ten, he is scarcely at all disconcerted; and with assurance concludes that the disease was much more serious then than now. This conclusion would have been legitimate had he left his patients to the unaided powers of nature; but he does not take into account his treatment, and he does not perceive that possibly the year in which he lost the greatest number of patients was that in which the mortality might have been least, had any other treatment been adopted.

On reading with attention what has been so beautifully written by Sydenham and Stoll upon the modifications necessitated in therapeutics by the epidemic constitutions which have been observed with so much care; there is produced, on the one hand, the conviction that the physicians who go on continually pursuing the same treatment, notwithstanding a change in the epidemic constitution, are men of narrow views; and, on the other hand, that there is a very great influence exerted by the epidemic constitution upon the action of the same medicines in diseases of which the local manifestations are the same.

You can now understand, Gentlemen, why in saying, at the beginning of this lecture, that the necessity, the utility even of bleeding in pneumonia, did not appear to me to have been clearly demonstrated, I took care to add, *in respect of the present time.* In fact, for years past, we have been traversing medical constitutions which do not necessitate recourse to that treatment, just as in the past, there have been medical constitutions which required it, and as there may be others in the future also demanding it.

So also, when Stoll, and still more when Rivière lauded the tartar emetic treatment, that treatment responded to the indications of a

then dominant medical constitution. For a long time past, that constitution has not shown itself, and the bilious symptoms, these which specially demand evacuant treatment, have not been recently observed.

Let me now resume consideration of the subject of *antimonials administered in large doses.*

To enable you to estimate correctly the immense difference which has been found to exist between their action as studied at different periods, it will be sufficient to glance at their immediate effects: and this will enable you to judge as to what may be their secondary influence. You will readily admit, that if it be possible to form an erroneous opinion regarding the secondary results of a particular treatment, there is never any room for mistake as to the immediate action. Though during a particular period, both in hospital and private practice, I could not prescribe for adults more than a gramme a day of the white oxide of antimony without exciting vomiting and diarrhœa—though during the same period I could not give in a day more than from 30 to 50 centigrammes of kermès, and that too, only when tolerance was secured—by administering along with it, a considerable dose of opium—though in fact, during the period I refer to, I was obliged to discontinue the use of the tartar emetic from the patients being unable to bear it, and from its always leading to serious symptoms—at another time, I have fearlessly given to an adult as his first dose 16 grammes of the white oxide of antimony in twenty-four hours, without the patient experiencing even the slightest nausea. I have at another time carried the dose of the kermès up to two or three grammes, without its being necessary to combine it with opium: and have without hesitation prescribed a gramme of tartar emetic, this large dose hardly inducing vomiting more than once or twice.

The immediate effects being so different, are we not justified in asking, whether the secondary effects do not vary as much? The fact ought to be recognised, that there is no ground for giving an absolute preference to any one of these preparations over the others; and it ought also to be understood that the dose in which they ought to be prescribed is subordinate to the influence of the prevailing medical constitution.

You perceive from what I have said, that the self-contradiction with which I am taunted in respect of the therapeutic properties of these preparations of antimony is more apparent than real.

Finally, that which seems to me at present to succeed best in the treatment of pneumonia—simple and perfectly uncomplicated pneumonia—is the contra-stimulant treatment, to use Rasori's expression—that is to say, the administration of antimonials, among which kermès ought to have the preference.

The efficacy of bleeding, I repeat, appears to me, for the present, very open to be disputed. In respect of *blisters*, the employment of which has been very general, from an impression that they greatly accelerate resolution of the inflammation, I concur with a large number of my professional brethren in thinking that, when the disease is at its height, they may increase the febrile excitement, and that when it is in a more advanced stage, they are useless. Moreover, during certain medical constitutions, a blister may be the starting point of very severe erysipelas.

It is, therefore, my practice to have recourse to kermès, or to kermès combined with digitalis. Not a week—indeed, I may say, not a day—passes, without your hearing me prescribe this medicine. Consequently, you are acquainted with my method of administering it.

To avoid a drawback incident to its use when prescribed in the form of potion, a drawback depending upon its locally irritant properties, its causing a pustular inflammation of the tongue, pharynx and œsophagus, similar to that produced by rubbing the skin with tartar emetic, I give it in pills. I order a mass to be made of kermès, extract of digitalis, and medicinal soap [*savon médicinal*[1]], dividing it so that each pill contains ten centigrammes of kermès and one centigramme of the extract of digitalis. The patient ought to take from ten to twenty-five of these pills during the day, at intervals as nearly equal as possible. When they produce vomiting and diarrhœa, I take care to give a drop of the laudanum of Sydenham with each pill, so as to establish a tolerance for the kermès. I continue this treatment during the whole of the acute period of the disease: when the febrile symptoms are subdued, I diminish the dose, but do not discontinue the medicine.

From my adopting this method of administration, you never see a pustular eruption produced by the kermès. Here, Gentlemen, we are brought into collision with the opinion of those who hold, with

[1] *Savon médicinal* is made of two parts (by weight) of oil of sweet almonds and one part of caustic alcali. Castile soap [*savon dur*], made of olive oil and soda, is also designated "savon médicinal."—Translator.

Laennec, that the appearance of pustules is indicative of saturation of the system with antimonials, just as salivation and mercurial stomatitis are the results of saturation, infection of the whole economy, with mercury. Supposing that this opinion, from which I dissent, were a real expression of the facts, you would obtain this saturation as quickly by administering the remedy in pills as in potion; and again, mercurial stomatitis is quite as apt to supervene after mercurial frictions or baths, as after the internal administration of mercury. I again repeat—what you can daily verify for yourselves—that antimonials given in pills, be the dose what it may, never produce inflammation of the mouth, pharynx and œsophagus, as they do in the form of potion, when they remain long in contact with the mucous membrane.

ERYSIPELATO-PHLEGMONOUS PNEUMONIA.

Gentlemen :—I place before you the lungs of a patient who died from a special form of pneumonia, which I have called *erisipelato-phlegmonous pneumonia*. Let me tell you why I have given this name to the disease. Generally, as you know, simple inflammation of the pulmonary parenchyma runs a course precisely similar to that of a boil—in this sense, that attacking a greater or less portion of the organ, it is at once that which it is destined to be, or, at least, it remains localised in the parts which it first seized, exactly as a boil in the cellular tissue is limited to its original situation. This form of simple pneumonia accomplishes its entire course of evolution, passing from the first to the second stage, and sometimes to the third : after which, resolution may take place, the patient recovering after having expectorated sputa to which pus imparts a characteristic aspect; or the pus may collect, forming a real abscess, which may burst suddenly into the bronchial tubes.

But the other form of pneumonia, which carried off the person whose autopsy we are now making, has not these simple characters. The parenchymatous phlegmasia, instead of remaining confined to the situation in which it is originally developed, has a peculiar tendency to invade other parts; it migrates like phlegmonous erysipelas of the cellular tissue.

In two words, here is what occurred in the case of our patient!—Ten days ago, he entered the clinical wards complaining of a violent

stitch in the right side, quite at the base of the chest. In the spittoon, we observed peripneumonic sputa of a slightly viscid character. The breathing was greatly oppressed, and there was high fever. Although these diagnostic data left no room for doubt as to the existence of pneumonia, I could not find, on auscultation, any physical sign of that disease. In no part, though the examination was made with the utmost care, could I hear râles or blowing. I then thought that it might be a case of pneumonia affecting a central portion of the lung, and foresaw that when the hepatisation reached the surface of the organ, there would be produced the stethoscopic phenomena previously in vain sought for. In fact, at my second visit, I heard fine crepitation, in front, about the tenth rib. From that time, no characteristic sign of the lesion was wanting.

On the following days, the physical signs indicated that the pulmonary inflammation was extending: it at first advanced to the middle of the axillary hollow, where it seemed to stop in its progress, there being at the same time observable a real amelioration in the totality of the patient's symptoms. The fever had abated, and he had even begun to feel some inclination for food, when the posterior part of the inferior lobe was attacked; and soon afterwards, the superior lobe became involved. The general symptoms, at the same time, became very severe: ataxic phenomena with delirium supervened: and the patient died.

Here then, Gentlemen, you have a pneumonia, apparently not at all serious at its commencement, very circumscribed, seeming on the first day to confine itself to a minute space, and even to show signs of incipient resolution:—you have this pneumonia all at once redeveloping itself with more than its original violence, and within nine or ten days invading progressively the entire lung, exactly as we see phlegmonous erysipelas, at first limited to the extremity of a limb, progressively attack the whole member, and give rise to most formidable symptoms.

This is one of the worst forms of pneumonia, one of the forms which baffles our means of treatment, in consequence of the constitution of the patient, exhausted by the successive shocks of the malady, being unable to respond to remedies otherwise most useful.

TREATMENT OF PNEUMONIA COMPLICATED WITH DELIRIUM, BY PREPARATIONS OF MUSK.

Musk not indicated in all cases of Pneumonia accompanied by Delirium.—Distinctions Essential to establish in relation to this point.

Gentlemen:—You have seen me prescribe musk a second time for a patient, occupying bed 24 of our ward for women, who had a relapse of pneumonia. I must state why I have done so; and explain to you the circumstances in which I consider this medicine to be useful.

In the first place, Gentlemen, let me remark that musk is a medicine which I seldom employ in the treatment of pneumonia. Many months will probably elapse before a case occurs in which its use is indicated; but rare though these cases be, as they may present themselves and greatly embarrass you, it is necessary to make you familiar with them. It is in the forms of pneumonia accompanied by delirium which were called *ataxic* and *malignant* by the old writers, that this treatment takes an important part. To Récamier belongs the credit of having in these later times assigned to it this honourable place.

What ought we to understand by the expression *ataxic pneumonia;* or, to speak more accurately, what is *ataxia in pneumonia?*

Nervous disorders, delirium in particular, supervening in the course of diseases are insufficient to constitute ataxia. To have an accurate understanding on this point, it is indispensable to distinguish several kinds of delirium in the pneumonia with the consideration of which we are now occupied.

In the first place, there is that delirium which is dependent upon the intensity of the peripneumonic fever, and which only indicates that the brain participates in the febrile excitement of the entire organism. It is not of common occurrence, except during the night, when the patients are in a drowsy state: it is, or may be, observed in all acute diseases accompanied by fever, and it has no special character. Musk certainly would not produce any beneficial effect upon this kind of delirium, inasmuch as it has no power over the inflammatory peripneumonic fever itself, and because the delirium will only yield to the means which stop the fever. It is also necessary to recollect that there is a form of delirium occurring in

persons of highly nervous temperament, which is not amenable to musk. It is well known that the persons who become delirious under the least febrile excitement are those in whom, *a fortiori*, inflammation of the lung excites very intense fever.

Secondly, there is delirium connected with suppuration of the pulmonary parenchyma, probably similar in kind to all forms of delirium produced by purulent infection, and which is of evil augury —"*a peripneumonia phrenitis malum*," to use the words of Hippocrates. It is always unpropitious, irrespective of the extent of the pneumonia. It is not amenable to musk.

Thirdly, there is delirium caused by one or several inflammatory complications situated in other parts than the lungs, and apt to be mistaken by the practitioner. It belongs to the first variety.

Fourthly, there is a delirium more dependent upon the malignity of the cause than upon the pneumonia itself. It is met with in pneumonia produced by poisoning, both in cases resulting from poisoning by articles of the materia medica or from morbific atmospheric miasmata, and by morbid poisons engendered within the economy. In all such cases, the pneumonia and the delirium are effects of the same cause. This is apparent in the pneumonia which complicates putrid fevers, acute glanders, &c. &c. and poisoning with acrid substances. In this class of cases, there is no indication for the use of musk.

Finally, there is a species of low delirium, attended by a want of harmony between the different symptoms, and a predominance of nervous phenomena bearing no evident relation to the inflammation of the lung. Under the influence of antiphlogistics or antimonials, this ataxic state increases. Were we to judge only by the diagnostic signs derived from stethoscopic and plessimetric examination, we should say that the pneumonia is not serious, and yet the vital power, prostrate and disorganised, collapses suddenly, and the patient dies. This is ataxia—this is malignity.

The characteristic of this species of delirium is the impossibility of associating it with any known material condition of the solids or fluids. In cases of this description, it would be a loss of time to seek for the cause.

This kind of ataxia shows itself, I repeat, by want of harmony between the local and general symptoms, and also by want of harmony between the different functional disorders, which ordinarily progress parallel with one another, or are correlatives. Let me explain myself.

An individual has a very slight attack of pneumonia: let us suppose that the disease is prevailing as an epidemic, so that a certain number of persons become affected in a manner similar to him. While in none of those seized, excepting the first mentioned, do nervous symptoms supervene, or at least if present, are proportionate to the extent of the lesion, in the individual first mentioned, there is delirium from the very first, without the inflammation having attained such a height as to justify one in supposing that the intensity of the inflammation is the cause of the nervous symptoms, and without the phlegmasia reaching the stage of suppuration, which if present, as I have just said, would explain the delirium. It thus becomes necessary to admit that in the individual first mentioned, there was a peculiar modality of the nervous system in virtue of which the nervous centres showed evidence of disorder not explainable by the slight local lesion. This is a point of the first importance.

In the second place, there is want of harmony, parallel or correlative, between the functional disorders. In pneumonia—in peripneumonic fever—proceeding regularly at the same time that the pulse becomes very quick, respiration becomes relatively accelerated. For example, while the pulse rises to 120 in the minute, the respirations are from 36 to 40: here, the disorders of respiration respond to those of the circulation. This is what takes place in ataxic pneumonia.

In the woman whose case is the subject of the present lecture, I insist upon this point, and beseech you not to forget it: while the pulse was 84 in the minute, the respiratory movements rose to 88. Respiration, consequently, had a frequency quite out of proportion to that which it generally bears to the arterial pulse. In place of being about one third slower than the pulse, it was quicker than the pulse. There was consequently a want of harmony between the functional disorders, which generally proceed in a parallel course.

It may also happen, Gentlemen, that the want of harmony which characterises ataxia is not so great in respect of the respiratory or circulatory functions viewed in relation to one another, as in respect of the two when compared with the nervous symptoms. Thus, along with the delirium, there may be no great frequency in the respirations, and—judging by the pulse and the temperature of the skin—the fever may be very moderate. Under what circumstances, and in what class of patients, is this peculiar form of delirium met with?

It is met with more frequently in women than in men, which is easily explained by the fact that disorder of the nervous system is more common in the former than in the latter. In men addicted to alcoholic liquors, or who drink stimulants to excess, it is also more usual than in others. In this class of patients, the nervous symptoms of which I speak occur not only in connection with an inflammatory affection such as pneumonia, but likewise as a consequence of severe traumatic lesions, such as a compound fracture of one of the extremities, a serious injury of a joint, or, it may be, after even a slight surgical operation. In telling you this, I am not stating anything which you have not already learned from your surgical teachers. Have you not heard them say a hundred times, that persons who have received wounds, have been surgically operated on, or have abused alcoholic liquors, are liable to a peculiar form of *delirium tremens?* Now, this delirium is analogous to the nervous symptoms to which I am at present directing your attention. It is liable to occur in the same persons in the course of a pneumonia, just as in the course of any other inflammation, or during a fever.

This *delirium of drunkards,* however, differs in its nature from the delirium which more specially characterises malignity. It is a purely nervous delirium: the brain is in a state of violent excitement: —the patients are restless, they wish to get out of bed, they talk nonsense with furious vivacity, just as if they were in the excitement stage of alcoholic intoxication; but there is no prostration of the vital powers as in ataxia.

If you employ musk to subdue the delirium of fever, or of suppurative pneumonia, if you employ it in cases in which the nervous symptoms depend upon the malignity of the cause which dominates the pulmonary inflammation itself, you mistake the indication, and your treatment is inevitably a failure. The consequences of your error will be disastrous. You will be prevented from recognising in musk the beneficial effects, which, when given in suitable cases, it is capable of producing; and will, therefore, not administer it in cases in which you ought to give it and obtain from it the best possible results.

Michael Sarcone proved the reality of these marvellous results, when, by the treatment I now commend, he checked delirium and disastrous excitability in some of his patients during the terrible Neapolitan epidemic of which he has left us a history remarkable from many points of view. He says:—

"When there was a threatening of delirium, and when in the aggregate of symptoms, there was a manifest sensitiveness, along with insomnia and great disorder of the nervous system, the only appropriate remedies were those which afforded quiet and repose to the patient. It is impossible to speak too strongly of the advantage obtained in these cases by the use of gentle calmatives and narcotics, when judiciously given. Musk was especially remarkable for its great efficacy in calming and subduing a tendency to convulsions which was dominant in a very marked degree in some of the patients. They at once fell into an agreeable and unhoped for torpor, and then by degrees passed into a state of repose, into drowsiness, and into sleep. The pulse acquired a more equal volume; and the respiration became less sighing. In cases in which delirium was not prevented, it was certainly less violent than had been threatened by the severity of the symptoms: and it never attained such dangerous manifestations in those to whom this drug was administered, as in other patients from whom it was withheld, or too long delayed, owing to I know not what prejudices."[1]

Gentlemen, let us have no misunderstanding on this point: I do not give musk indiscriminately in the delirium of pneumonia any more than I do so, in that of scarlatina or small-pox: I only give it in that peculiar form, which, manifesting itself in maladies characterised by nervous disorder, are yet not of a serious nature. In these cases, musk becomes a sort of regulator of the nervous system, which then responds in a regular manner to the assaults of the disease.

What occurred in the case of our patient of bed 24 St. Bernard's ward? From the second day of her pneumonia, this woman was delirious, though the local affection remained very limited in situation, and did not pass beyond the second degree. The respirations rose to 88 in the minute, although the pulse was only 84. The ataxia was evident: the indication for giving musk was precise. Whilst I administered it, however, I did not discontinue giving the kermès. With kermès I combated the inflammation, while I at the same time directed the antispasmodic remedy against the nervous element of the disease.

You have seen the results of this treatment. No doubt, on auscultating the chest, you have satisfied yourselves that it has not

[1] Sarcone:—Histoire des Maladies Observées à Naples, T. II, p. 240.

stopped in any degree the inflammation of the lung. I made no such pretension; for, treating the case by antimonials, or by bleedings, the indications for employing which are, as I have already said subordinate to the dominant medical constitution, we may conduct the pneumonia to its termination in resolution, but we cannot cut it short in twenty-four, thirty-six, or forty-eight hours, as has been supposed possible by some physicians. I therefore waited to see the local affection run its course; but I also waited to see the cessation of the nervous symptoms. The respirations fell from 88 to 44 in the minute, although the pulmonary lesion being a little more extensive than before, one might on the contrary have expected acceleration of the breathing, had that been dependent upon the state of the lung. Although the number of the respirations had not yet come down to the normal standard, there was every reason to hope that this diminution would take place on the following day.

The very violent delirium, which might have caused anxiety, had calmed down: during the night there was only a little restlessness, and in the morning, the patient answered questions with precision. The musk had induced this sedative effect, though I had not had occasion to give more than 50 centigrammes in the twenty-four hours. I still continue to use it. As the patient has been entirely without sleep during the last three days, and as this insomnia is a phenomenon pertaining to ataxia, I shall, if it continue, combine small doses of opium with the musk, or I shall give the opium by itself. The combination of musk and opium is also recommended by Sarcone, when there is exhausting obstinate sleeplessness in addition to the other nervous symptoms.

But it is not enough to be able to recognise the indications for giving musk in pneumonia accompanied by delirium—there are certain rules connected with its administration, which it is indispensable to know. It may be prescribed to the extent of a gramme a day divided into ten doses given in the form of pills, one pill being taken every hour till there is a remission in the ataxic symptoms, which generally occurs within eight or ten hours. At the end of that time, according to Récamier, if no benefit has been obtained, we need not look for results, as they are either obtained promptly or not at all.

I have still a word to say, in conclusion, regarding our patient. Her pneumonia is not extensive, and the reactional symptoms, separated from the nervous symptoms, also indicate that the case is

not serious. Under the circumstances, therefore, I consider that in this case recovery will take place.[1]

Gentlemen, I think it very important to bring these points under your notice, and to state with precision the indications for the treatment which you have seen me employ, because I have often heard its efficacy called in question by very worthy persons, who have unsuccessfully employed musk in pneumonia with delirium. Their failure did not depend on the remedy being bad, but on its having been given in unsuitable cases, in very different forms of delirium from that now under our consideration. By such errors in diagnosis, the character of the best therapeutic agents is compromised. When you confound with one another the different phenomena which may supervene in the course of a disease, you inevitably fail, through attacking symptoms which are not the same by the same remedy. The remedy having failed, from its employment not having been indicated, you cannot see its utility, and you deprive yourselves of a powerful agent which in appropriate circumstances is of real service.

PNEUMONIA OF THE SUMMIT.

Not necessarily accompanied by Delirium.—Delirium may also occur in Pneumonia situated in the Centre or Base of a Lobe.—Pneumonia of the Summit is not necessarily more dangerous nor more tedious; but this statement requires limitation in respect of Tuberculous Patients.

Gentlemen:—In beds 4 and 18 of St. Agnes's ward, you have seen two men with acute simple pneumonia. Both these men, who are of good constitution, in the prime of life and under thirty, took the disease, which brought them to the hospital, in the way which is most usual, that is to say, in consequence of a chill. In both, there was this peculiarity, that the pneumonia occupied the summit of the lung. Both recovered completely and rapidly: no complication arose to impede the cure. Let me briefly recapitulate the facts.

The first subject had been ill for seven days: a violent shivering fit and a stitch in the side announced the beginning of the morbid

[1] After having been under treatment for some days, this woman made a complete recovery, and was able to leave the hospital.

symptoms: almost immediately afterwards, there supervened cough accompanied by expectoration. The fever which forthwith declared itself has continued ever since. When this patient came into hospital on the third day of the disease, we found him expectorating characteristic sputa of a saffron yellow colour, aërated, viscid, adherent to the vessel, and leaving no room for doubt as to the diagnosis. The harshness of the sound elicited on percussing the left side of the chest over the infra-spinous fossa of the scapula and under the clavicle, the crepitant râles and the blowing sound of expiration perceived on auscultation of these regions, confirmed the view already satisfactorily arrived at, as to the nature of the case, from an inspection of the sputa and the symptoms complained of by the patient. We had pneumonia; and that pneumonia was situated in the summit of the left lung.

Next day, the blowing expiratory sound had given place to tubal blowing, and numerous puffs of fine crepitation were heard throughout a space more extended than on the previous evening. On the sixth day, the stethoscopic signs were still more decided.

Contrary to what I have frequently observed this year, the patient complained of obstinate constipation, although he had been taking kermès in large daily doses. I found it necessary to give him two calomel pills, each pill containing five centigrammes; and in addition to this, two grammes of powder of jalap. These remedies produced the desired effects.

This morning—the eighth from the beginning of the malady—I find the patient without fever, the skin is in a good state, the pulse is full and not quick, its amplitude being proportionate to the constitution and strength of the subject. The stethoscopic signs are modified; and we now hear the vesicular murmur, accompanied it is true by fine subcrepitant mucous râles, in situations, where, forty-eight hours previously, we heard tubal blowing and crepitant râles.

The resolution of the pneumonia has, therefore, fairly begun. Yesterday, however, a circumstance arrested my attention, although the condition of the patient seemed satisfactory. I refer to the character of the sputa. The saffron colour which they presented during the first days of the illness had become of a deeper shade; and yesterday, though still retaining their viscid character, they had assumed the aspect of wine lees, or prune juice, generally an evil omen. As, however, the sputa were still viscid, I was less alarmed by their change of colour. In point of fact, it is not so much the

sputa assuming the prune juice colour in peripneumonia, as their ceasing to be viscid, and assuming a peculiar diffluent character, which constitutes the unfavourable prognostic. This morning, we observed that the sputa had reassumed a slight saffron tint, and were not profuse.

The history of our second patient is nearly identical with that of the first. His peripneumonia, contracted under similar circumstances, occupied the same situation, ran the same course, and likewise terminated as rapidly in recovery.

In both cases, my treatment was the same: I employed antimonials: I had recourse to kermès.

These cases form an appropriate sequel to what I have been saying on the use of musk in pneumonia. In fact, Gentlemen, the form of delirium so remarkably subdued by that remedy, is perhaps most frequently met with in pneumonia of the summit. The reason of this I cannot tell; but the fact is generally admitted. It is evident, however, that pneumonia of the summit does not necessarily induce nervous symptoms; as is proved by the cases of the two men of St. Agnes's ward. You also learn from these two cases that pneumonia of the summit is not inevitably more serious than pneumonia of the base.

I do not deny that in persons of the tuberculous diathesis, pneumonia of the summit is a more serious affection than in those not under that diathesic influence. This does not depend upon the pneumonia itself, but upon the risk there is that its presence may hasten the development of phthisis, by calling forth the manifestation of the diathesis by accelerating the evolution of tuberculous products in their favourite seat—the summit of the lung. With this limitation, I maintain that pneumonia of the summit is not more dangerous than pneumonia of the base or centre. The gravity of the inflammation does not depend upon its situation, but upon its extent and nature. With regard to *extent*:—a pneumonia which simultaneously invades an entire lung is, if other conditions are equal, more grave than inflammation limited to one lobe, and double is always more dangerous than single pneumonia. With regard to *nature*:—the relative gravity, I should say, depends on the specialty of the nature, which varies with the epidemic constitution, the previous condition of the patient, as well as with certain other influences, an intimate acquaintance with which we are unable to obtain, and which we know only by their effects.

LECTURE XXXVII.

PARACENTESIS OF THE PERICARDIUM.

Cases.—Historical Summary.—Harmlessness of Tapping the Pericardium and injecting Solutions of Iodine.—Better to make the opening with the Bistoury than with the Trocar.—Dropsy of the Pericardium almost always associated with some other diseased state, particularly with the Tuberculous Diathesis.—Paracentesis affords relief and prolongs life placed in immediate jeopardy.

Gentlemen:—In your presence, I performed paracentesis of the pericardium upon a patient who lay in bed 2 of St. Agnes's ward. The man died five days after the operation; and I placed before you the morbid structures found at the autopsy.

This patient was a young man of 27, who came into our clinical wards on the 2nd June 1856: he dated the commencement of his illness from a few days prior to his coming into hospital. I observed at my first visit, that there was great oppression of the breathing: throughout nearly the whole of the chest, I heard sibilant, mucous, and subcrepitant râles—in fact, all the signs of capillary bronchitis. The intensity of the fever quite corresponded with the severity of the local symptoms. This young man told us that two years previously he had had a severe pulmonary affection for which he had taken cod liver oil.

I prescribed preparations of antimony and digitalis: I administered purgatives: speedy improvement was the result.

I did not, however, take into account the persistence of the fever, still less the persistence of the peculiar anxiety experienced by the patient. Upon afterwards carefully examining the heart, I heard a blowing sound; and also a friction sound accompanying both sounds of the heart, a little more marked at the apex, which is not usual in pericarditis, in that disease the double friction sounds being heard around the base of the heart: in other respects, the pulsations were perfectly distinct.

This young man had never had rheumatism.

My opinion was that there existed endocarditis, and a lesion of the mitral valve complicating the pulmonary catarrh.

Once my attention was called to this point, I carefully auscultated the heart every day. A week had scarcely elapsed, when the stethoscopic phenomena presented a strange modification. I distinctly heard a double bellows sound at the apex, and some days later, there was a reduplication of the second sound, so as to constitute a third sound, called the *bruit de rappel* or *bruit de galop*.

The precordial dulness increased greatly, the blowing sounds became more and more distant from the ear, and at last were hardly audible. The pulsations of the heart became very obscure, and then ceased to be heard. The pulse at the wrist continued to be rapid, but it was regular, and of fair strength.

There was no doubt as to the existence of the pericarditis, the progress of which I followed. The arching of the precordial region, the limits of the dulness—extending to the right beyond the median line, and reaching on the left to two or three centimeters external to the nipple, descending as far down as the diaphragm, ascending to the third rib, thus circumscribing a space of about 20 centimeters—clearly indicated the existence of extensive effusion into the pericardium.

The anxiety of the patient increased proportionately to the increase of the pericardial dropsy, although in respect of the cough, there was a great change for the better.

No amelioration of symptoms was obtained by the administration of digitalis, calomel and purgatives, and the application of blisters to the region of the heart.

Matters had been going on in this way, or getting worse, for six weeks, when I perceived œdema of the extremities, puffiness of the face, and great paleness of the skin. These symptoms suggested that there was probably albuminuria; but upon analysis of the urine it was found not to contain any albumen. I then concluded that the anasarca, oppression, and anxiety were all dependent upon obstructed circulation. The pulse had become small and very rapid.

Under the circumstances, I considered that paracentesis of the pericardium was indicated. I nevertheless allowed a fortnight to elapse; for although two years previously, in my wards, I had operated successfully in a similar case, I hesitated to have recourse to an operation which one never without trembling decides upon

performing. The symptoms, however, became so urgent, and death appeared so imminent, that I determined to delay no longer. On the 1st August, I invited my colleagues of the Hôtel-Dieu to meet me in consultation on the case.

I submitted to my colleagues the triple question of diagnosis, prognosis, and treatment. They were all of opinion that there was pericarditis with effusion, which was estimated at less than a litre. They all thought, that, looking to the anxiety of the patient, the general puffiness, and the extreme paleness of the tissues, death would occur within a few days. They likewise all thought, that although the operation offered but little chance of success, that the treatment which afforded the greatest chance was the prompt evacuation of the fluid by tapping.

Paracentesis having been decided on, I forthwith proceeded to perform it. I employed a bistoury in opening into the chest. The incision was made in the centre of the circumference marked out by dulness, below the nipple, and in the nearest intercostal space. After cutting in succession, with the utmost caution, the skin and muscles, I reached the pleura. This membrane was next cut through. Upon introducing my finger into the cavity of the chest, I encountered resistance from the distended pericardium. I did not feel the heart beating under my finger. I then cut through the successive layers of tissue, separating them by means of a grooved director. At last, the point of the bistoury having penetrated a little too far, some slightly red serosity spurted along the blade. Using the grooved director, I enlarged the incision only to the extent of half a centimeter: a gush of similar fluid then issued from the wound, and in part spread under the layers of tissue: nearly 100 grammes were collected in a pallet. This fluid immediately coagulated like currant jelly: the flow then ceased. I introduced several gum elastic sounds, but by so doing did not succeed in obtaining any more fluid. By causing the patient to be placed on his left side nearly 200 grammes of yellow coloured serosity issued from the opening: this, therefore, was very different from the first-drawn serosity, which, when received in the same pallet, coagulated but imperfectly, and contrasted by its amber colour with the appearance of red currant jelly presented by the other.

Gentlemen, it was seen at the autopsy, that one of these fluids must have come from the pericardium and the other from the pleura.

From the cessation of the flow, I thought that the pericardium was occupied by false membranes, which retained the fluid within enclosures. I tried to inject a solution of iodine, but none of it passed into the cavity of the pericardium: perhaps about a tablespoonful penetrated into the pleura. The wound was then closed by means of diachylum plaster.

Notwithstanding the small quantity of fluid withdrawn, certainly not more than 400 grammes, including both that which came from the pericardium and the pleura, the patient was decidedly relieved by the operation. The pulse became slower and fuller.

Some air entered the chest during the operation; and mixing with the serous fluid which was withdrawn, gave it a frothy appearance. On auscultating the patient after the dressing, we heard the sounds of the heart unaccompanied by the mill-wheel sound [*bruit de roue de moulin*], which has been given as a characteristic sign of hydro-pneumo-pericardial disease.

The young man, who formerly could only lie on the left side, now found that he was most comfortable when on the right side. Matters went on pretty well till the afternoon: but when M. Beylard and I saw the patient about four o'clock, we found him in a very excited state, with high fever, and a pulse of 124. About three hours after our visit, he was suddenly seized with an attack of eclampsia: the convulsions were confined to the right side of the body. During the night, the attacks recurred every half-hour.

On the following morning, the right side of the body and the tongue were completely paralysed; but, strange to say! during the attacks which I witnessed, consciousness remained to a certain extent. The patient tried to answer questions; and with the left hand he pressed the convulsed muscles of the right cheek, to restrain their disorderly movements.

Gentlemen, before I go any further, let me try to explain these attacks of eclampsia. Ought they to be attributed to the operation? Let us for the present leave out of view the question of the advisability of the operation—a question resolved in the affirmative by my colleagues in the Hôtel-Dieu and myself. Let it be granted that surgical interference was as rash a proceeding as could have been adopted, still, it was not one of those great surgical operations —those serious traumatisms—which sometimes induce nervous symptoms.

Again, in the convulsions of our young man, there was nothing

like the phenomena of tetanus. Let it be granted that the paracentesis increased the intensity of the inflammation of the pericardium: but then, do inflammations of the serous membranes, be they ever so violent, or whatever may be the extent of the membranes involved—do they usually lead to such symptoms? The answer is obtained by an appeal to clinical facts. Never does the most violent pleurisy, never does the most acute peritonitis following perforation or strangulation of the intestine, induce convulsions—not at least in adults. Have you ever heard that it was otherwise in pericarditis?

The circumstances in which the patient was placed gave us a better explanation of his symptoms. You are aware that general anasarca, even when there is no albuminuria, produces a peculiar predisposition to eclampsia. This is observed principally in pregnant women and in children. A feverish attack or a mental emotion may be the immediately exciting cause. You know also how much an anæmic condition conduces to the occurrence of convulsions. In our young man, two predisposing causes, anasarca and anæmia, existing in a high degree, it is not surprising that mental emotion occasioned by the dread of the operation—for he was much alarmed by the meeting in consultation held around him—should have induced the nervous symptoms of which I speak.

I prescribed preparations of musk and valerian; and for the time, they seemed to subdue the symptoms. I was beginning to hope that matters were taking a favourable turn, and no new heart symptoms had occurred, when the respiratory organs were again attacked. On August 4th, the fourth day after the operation, I observed more oppression of the breathing, as well as some cough accompanied by profuse expectoration of slightly viscid matter; and I heard subcrepitant râles. Next morning, these symptoms had become still more alarming, and caused me much more anxiety than those referable to the heart.

From the very weak state of the patient, I was unable to examine the chest as carefully as I should have wished. However, the dulness of the precordial region indicated that there was still a very considerable amount of effusion, though much less than before the tapping. The patient died on the evening of Tuesday 5th March, five days after the operation.

The autopsy was performed with the greatest possible care. The ribs on the left side, from the axilla to the base of the chest, were sawn

through: on the right, the sternum was separated from the costal cartilages, then the sternum and anterior part of the chest were detached in such a way as to remove the trachea, the lungs, and the heart intact within the pericardium. In the left pleura, I found a citrine coloured fluid similar to that which flowed during the second period of the operation. There were neither false membranes, fibrinous floculi, nor adhesions.

The pericardium was reddish in colour, and resembled an enormous globe as large as a man's head. It had no adhesions with the ribs, and was only in its upper part covered by a thin portion of the left lung, which was firmly attached to it by an adhesion of old date. Situated in the anterior mediastinum, which it had separated, it resembled a fruit planted behind the sternum on a large base, and floating in the pleural cavity which it had opened. Almost opposite the point at which the opening had been made in the intercostal space, a violet spot was observed on the inside of the pericardium—the mark left by the cut of the bistoury. The blunt end of a probe entered it easily. The false membrane which lined the serous membrane in the situation of this opening was red, apparently from recent sanguineous effusion. On opening the pericardium, there flowed out nearly a litre of a reddish fluid, identical with that collected in the pallet during the first part of the operation. A very few fibrinous floculi were observed floating in this fluid.

The heart was at the bottom of this sac, at least ten centimeters from the wall of the cyst, and from the point where the puncture had been made. It was covered by a thick, reticular, dirty-yellow false membrane, as was likewise the whole of the interior of the sac. The thickness of the wall of the cyst might be about five millimeters.

On cutting the heart, it was found that below the serous coat there was a thick lardaceous cellular tissue, resembling a layer of fat.

The heart was of rather more than the natural volume: but the hypertrophy was concentric, the cavities being narrower than normal. The flap of the valve was supple, thin, and without appreciable change of structure. The orifices, however, allowed the finger to pass less easily than in a healthy heart.

In the lungs and bronchial glands, there were disseminated both crude and softening tubercles. Near the pancreas, there was a mass of softened tuberculous glands. The mesenteric glands were engorged. There were some intestinal adhesions.

In the encephalon, the only morbid appearance seen was a little softening of the falx cerebelli. It ought, however, to be stated, that the autopsy was made during very hot weather.

The *post-mortem* examination gave us complete information as to what took place during life, and during the operation. There was pleural as well as pericardiac effusion. The stoppage in the flow of the pericardiac fluid was produced by a mechanical cause which is easily explained. Once I had penetrated the pleura, I was afraid to go farther, and consequently only made a very small opening in the pericardium opposite the opening in the thoracic walls. The parallelism between the two openings was soon destroyed, and the result was the flow of the pericardiac effusion into the pleural cavity.

Could I have avoided this accident by at once introducing a sound into the pericardium through the opening made by the bistoury, or by puncturing with a trocar so as to enable the fluid to flow through the canula? But even then, there would have been difficulty in avoiding the inconvenience which arose from the fluid oozing out between the lips of the wound and the sides of the canula: as soon as the canula was withdrawn, the parts resumed the vicious position which it was supposed could have been prevented.

When I come to discuss the mode of performing paracentesis of the pericardium, I shall return to this question, to explain how it is that the inconvenience referred to is, in my opinion, a matter of small importance. Then, I shall also tell you, why I prefer to operate with the bistoury, and not with the trocar employed by others, and in particular as you have seen it used even in my wards, in the case regarding which I have now been speaking. Finally, I shall have to tell you that I reject Riolan's operation, adopted by Skielderup and recommended by Laennec, which, consisting in penetrating into the pericardium by trepanning the sternum, is a proceeding which seems to me at the least to be useless.

Gentlemen, as I have just mentioned, I have had occasion to perform paracentesis of the pericardium in the case of a young man in our wards. This first case (published by my friend Dr. Lasègue and me in the *Archives Générales de Médecine* for November, 1854) might have been considered as encouraging.

The patient, a lad of sixteen, was admitted to St. Agnes's ward on the 2nd February 1854. He was pale and weak; but he declared that he had never had any serious illness. Four or five days before he came into the hospital, he had suffered from very severe frontal

headache, which was soon followed by extreme lassitude and pain in the precordial region.

On admission, he had intense dyspnœa: his pulse was 150: he had a little cough: and there was an expression of suffering in his countenance. On percussion, I found great dulness in the region of the heart, reaching as high up as the second rib, and extending from the right margin of the sternum to very far forward in the left side of the chest, in which direction, however, its limits could not be exactly defined: posteriorly, the left was less sonorous than the right side of the chest: the sound of the pulsations of the heart was obscure and distant. The patient had never completely fainted; but had a constant feeling that syncope was imminent. I ordered a large blister to be applied to the precordial region; and prescribed an infusion of digitalis as a tisane.

During the month of February, while the pulse continued very rapid, the pulsations of the heart were better heard at intervals, becoming, however, indistinct at other times. The dulness in the precordial region occupied almost uninterruptedly a space extending upwards and downwards of seventeen centimeters, and transversely of eighteen centimeters: during two days only—from the 18th to the 20th—did it appear to diminish. At this time, there was a double cardiac friction sound, which was most appreciable at the base of the heart. The arching of the precordial region became more and more marked on the left side of the chest: the signs of pleural effusion became more and more distinct, there being posteriorly, below the scapula, egophony and a bellows-sound.

On the 17th March, it was noted that the arching had been greater for eight days, and that profuse diarrhœa had supervened. The patient was losing strength and flesh, and could no longer move in bed without inducing a tendency to faint. The face was pale and livid: the oppression of breathing was extreme: respiration was short and sighing: percussion, and even the simple application of the hand over the precordial region, produced pain and pang: the pulse, small and feeble, was 120: the dulness extended up to the clavicle.

Next day, in consequence of the symptoms having become more serious, and death threatening, I resolved to have recourse to paracentesis of the pericardium. Professor Jobert (of Lamballe) operated. In the fifth intercostal space, at about three centimeters from the left margin of the sternum, he made an incision through the skin and cellular tissue to the intercostal muscles. He then

introduced slowly, steadily by continuous pressure, obliquely from right to left, a trocar provided with a piece of membranous material. On withdrawing the stem of the instrument, some drops of reddish serosity flowed through the canula. When the canula was left free in the wound, it was moved about by the action of the heart, being raised up by each contraction of the organ.

During the operation, the patient (who had requested its performance but was alarmed by the preparations) was pale, and groaned. His pulse, very slow and almost imperceptible, ere long regained its usual strength and quickness. He had no feelings of general discomfort, no great amount of oppression, and no faintness.

At first, the fluid flowed pretty freely, although it did not spurt out in a jet. When about sixty grammes had been collected, the flow become slower: the canula was then fixed in the wound, whereupon, without favouring the flow by any manœuvre, the fluid dribbled out. The operation was performed at nine in the morning: at half past nine, the patient stated that he neither experienced any relief, nor felt increased oppression. The canula was removed at half-past ten: by that time, the flow amounted to 400 grammes. About noon, the young man felt a little better. At my evening visit, he expressed himself as having obtained great relief. He breathed quietly. His pulse was 134, and full. The dulness only extended upwards to within four finger breadths below the clavicle, and did not go more than two centimeters to the right of the middle of the sternum: on the left, it extended to a line drawn vertically downwards from the anterior boundary of the axilla. The pulsations of the heart were much more distinct; and the apex of the heart was appreciably raised.

Forty-eight hours later, the report made was to the following effect:—the ameliorated condition is maintained: there is increased —almost tympanitic—resonance anteriorly in the left summit: the respiratory murmur is audible from the clavicle to the fourth rib, where dulness commences: the sounds of the heart are becoming more and more distinct: on the left side, posteriorly, there is dulness, bellows-sound, and egophony: there is little cough, and almost no oppression of the breathing; there is not much fever.

On the 22nd March, the pleuritic effusion was progressing: the heart was pushed over to the right side, and there was great distension of the left side of the chest. The patient lay on the right side, and complained of an exceedingly painful stitch.

Up to the end of the month, the pleuritic effusion went on increasing; the fever augmented; and the cough, more frequent, was accompanied by the expectoration of thick white matter. Mucous râles were heard at the summit of the left lung. The oppression of the breathing had become more decided, but still it was not nearly so urgent as it had been before the operation. There was some diarrhœa, which was moderated by the administration of nitrate of silver.

On the 30th, seeing that there was effusion occupying the entire left pleura of an individual already exhausted by his disease, I did not hesitate to perform paracentesis of the chest. A first opening made in the sixth intercostal space, in the axilla, did not afford exit to one drop of fluid: the trocar had been arrested by a very tough false membrane. A second opening, made a little more posteriorly, and lower down, allowed about 500 grammes of fluid to flow out. The operation was not followed by any particular occurrence, and did not occasion any untoward symptoms.

On the 2nd April, some subcrepitant râles were heard in the lower part of the left lung; but there was neither blowing sound, egophony, nor any appreciable embarrassment of breathing. The diarrhœa continued. The patient had insomnia.

From the first days of April, up to the 28th May, the day on which the patient left the hospital, there was no reproduction of the pleuritic or pericardiac effusion. The pulsations of the heart could be felt by the hand: the sounds of the heart were not accompanied by any blowing or friction sounds. The extent of dulness was greater than in the normal state: the arching was quite obliterated; respiration was fairly free, and the young man made no complaint of dyspnœa. He sat up in bed to play, had appetite for food, and declared that he was very much pleased with his improved position.

His general state, however, was far from satisfactory: his cough was more frequent: he had recurrence of the fever, particularly towards evening: the diarrhœa, more moderate it is true, was not yet checked: and he did not regain strength, notwithstanding the use of medicinal tonics and a strengthening regimen.

As he was weary of the hospital, and attributed the slowness of his convalescence to his remaining so long, he asked to be allowed to leave. The signs of tuberculisation, which had been becoming more evident for a month, were quite decided at the date of his leaving the hospital. The symptoms then noted were the fol-

lowing:—at the summit of the left lung, anteriorly, there were observed dulness, sibilant râles, gurgling during forcible inspiration, and an absence of the bellows-sound; posteriorly, at the summit, there existed subcrepitant râles, and at the lower parts of the lung there were mucous râles. On the right side, respiration was puerile in front: behind, expiration was blowing, the voice resonant, the râles dry and sonorous, and the pulsations of the heart were strong and distinct. The fits of coughing recurred unaccompanied by any special expectoration. The patient had dyspnœa, obstinate diarrhœa, emaciation, and hippocratic deformity of the fingers.

Notwithstanding his feeble state, he was able to be taken home in a carriage to his family, in the department of Eure-et-Loir. During the first fortnight of June, we heard that his state continued very much as when he left the hospital. Since then I do not know what has become of him.

This case, Gentlemen, as I have already said, is an encouragement to perform paracentesis of the pericardium, because it is an example of the operation preventing death otherwise imminent.

In one of my recent lectures, when giving you a rapid historical sketch of paracentesis of the chest in pleuritic effusions, I showed you that although the indications for operating had not been formulated with precision, the operation had been performed at different periods anterior to our own. Long before any one ventured to perform it, it had been considered from a theoretical point of view, and supported by sound arguments, as a proceeding likely to be useful and quite free from danger. It was not so in respect of paracentesis of the pericardium. A long period elapsed before it was advocated on speculative grounds; and while some expressed an opinion that it might be possible to operate for effusions into the pericardium as had been done successfully for effusions into the pleura, the proposal to bring a cutting instrument near so delicate an organ as the heart was rejected as inexcusably rash.

To Senac is generally attributed the honour of having pointed out the possibility of successful paracentesis of the pericardium;[1] but a century previously, Riolan had formulated the indications for resorting to that operation.[2] Senac certainly never performed the operation, though some compilers have made a statement to the

[1] Senac:—Traité de la Structure du Cœur et de Ses Maladies. Paris, 1749.

[2] Riolan:—Enchiridion Anat. Lib. III. Lugduni Batavorum, 1649.

opposite effect. Several authors cited by Sprengel, taught that the operation ought to be tried, as, if left to itself, dropsy of the pericardium must prove fatal; but they did not venture to set the example of performing it. Ricter, while he admitted its utility exclaimed:—"*Intrepido opus est animo ad talem operationem instituendam;*" and Van Swieten, who was not over-timid in employing remedies of risk in extreme cases, does not speak more confidently: he says—"*Quam audax facinus debet videri omnibus si quis cogitaret de pertundendo pericardio dum hydrope turget.*" However, notwithstanding the difficulties with which the diagnosis of dropsy of the pericardium was surrounded, although experience had not yet pronounced its decision, he recognised that it was allowable, rather than leave the patient to the cruel alternative of death, to afford by operation an outlet for the effused fluid:—his words are—"*Interim generale axioma practicum omnibus probatur: tentandum esse potius anceps remedium quam nullum, dam certa pernicies imminet.*" He concludes his remarks by describing the manner in which the operation ought to be performed.

About the same period, Benjamin Bell, Camper, Arneman, and Conradi all recommended the operation to be tried, while they proposed different methods of operating, to the consideration of which I shall afterwards return:[1] the counsels which they give are, however, purely theoretical. At a later date, Desault, who originally regarded paracentesis of the pericardium as almost impracticable on account of the difficulty of determining the indications, found a case in which he thought he had a good opportunity of carrying out practically the precepts of his contemporaries: but his first operative attempt was far from being a success. The effusion in fact, which he had desired to evacuate by tapping was not contained in the pericardium, as appears by his account of the case, which is given with the most perfect simplicity and candour.[2]

This case then, usually quoted as the first in which paracentesis of the pericardium was performed, has no claim to figure in the history of that operation, and cannot be appealed to, except as an argument to show the obscurity of the diagnosis. Larrey's case is not more conclusive, although it has also been often appealed to.[3]

[1] Van Swieten:—Comment. in Aphorismos Boerhaavii. Parisiis: T. IV p. 122.

[2] Desault:—Œuvres Chirurgicales recueillies par Bichat. T. II. 1798.

[3] Larrey:—Sur une Blessure du Péricarde suivie d'Hydro-péricarde. [*Bulletin des Sciences Médicales:* 1810.]

These two cases of alleged paracentesis of the pericardium, both originating in errors of diagnosis and both terminating in death, hardly advanced the question: they may be considered as leaving matters as they were. With the exception of an interesting essay in which Skielderup defends tapping the pericardium, without, however, citing cases in support of his views,[1] the operation was either forgotten or very severely criticised.

Corvisart,[2] who in the first edition of his work, which appeared in 1806, was satisfied to report Desault's case, proposed paracentesis of the pericardium in a subsequent edition published in 1818: he recommended an incision with the bistoury, as preferable to a puncture with the trocar. He stated, however, that he thought the possible advantages of the operation would rarely counterbalance the danger to which it exposed the patient.

The opinion then held in Germany was similar to that of Corvisart: and Kreysig, in a work which he published in Berlin in 1816, on paracentesis of the pericardium, holds that it would be very difficult to apply the operation usefully in practice. Besides, he said, the disease being of such a nature as to render tapping of little avail, there is always the fear of consecutive inflammation of the heart with its inevitable results. The introduction of air would induce suppuration leading to death. In harmony with the opinion of his contemporaries, he added, that the means of diagnosis were not sufficient to excuse the temerity of resorting to such an operation.

In France, Laennec, adopting the views of Senac, thought that there might be a possibility of curing dropsy of the pericardium by a surgical operation. He only supported this opinion, however, by presumptions.

Richerand went farther than Laennec, by proposing as a means of radical cure, a treatment by astringent injections similar to that pursued in dropsy of the tunica vaginalis. Such was the position of the question as one of science, when, in 1839, Schuh, one of the principal physicians of Vienna published a remarkable work entitled:—"*De l'Influence que la Percussion et l'Auscultation sont appelées à exercer sur la Pratique Chirurgicale.*" He therein

[1] Skielderup:—De Trepanatione ossis Sterni, et Apertura Pericardii. [*Acta Nova Societatis Medicinæ Hafniensis:* 1818.]

[2] Corvisart:—Maladies et Lésions Organiques du Cœur et des Gros Vaisseaux. Paris: 1806.

reviewed the services rendered by both the new means of diagnosis, and specially applied himself to show the reliability of surgical diagnosis based upon signs which were almost certainties. Pleuritic effusions, for the treatment of which he invented (as I have told you) a special apparatus, and pericardiac effusions were cited as the most convincing examples in support of his opinion; and he summed up by stating that, suitable cases presenting, he should not hesitate to perform one or other of these pararcenteses as the case might require. An opportunity soon presented itself. In the following year, paracentesis of the pericardium was practised for the first time. The operation was performed in the wards of Professor Skoda, and Schuh was the operator.

Notwithstanding the interest which attaches to the case published by Skoda, and which Dr. Lasègue and I have reported *in extenso*, in the paper which we contributed to the *Archives Générales de Médecine*, it would be occupying your time unnecessarily to repeat the details upon the present occasion; particularly, because when considered from the special point of view now engaging our attention, this first attempt at paracentesis of the pericardium was not very encouraging. In fact, upon two occasions, the attempt to make the puncture was a failure. On the first occasion, the instrument penetrated a heterologous mass six inches thick occupying the mediastinum: this mass impinged upon and altered the sternum: the inner surface of the clavicle, and upper four ribs were attached to the vertebræ, adhered strongly to the lungs, and encircled the large vessels and the windpipe. From this first puncture, there came only a very small quantity of sanguinolent serosity of the consistence of syrup. After prolonged and ineffectual attempts to obtain more fluid, the canula was withdrawn; and it was resolved that a second puncture should be made in the intercostal space immediately under that first perforated. A certain quantity of reddish serosity was obtained by this second puncture; but the relief which the patient experienced was of short duration. Nevertheless, this case afforded valuable instruction; for it was a decisive experiment to show that paracentesis of the pericardium ought not to be looked upon as one of those bold proceedings which are hardly justified even by success.

In the following year, Dr. Heger, a pupil of the learned professor of Vienna, operated in a case of dropsy of the pericardium, which though not complicated with conditions so unusual as those met with

in the former case, was associated with other pathological states, which, as I shall forthwith tell you, and as Dr. Aran has shown generally coexist with this disease.

Dr. Heger's case published in a German journal deserves to be reported to you.

The patient was a shoemaker aged nineteen. He stated that on admission to the hospital he had had the disease for about six weeks which brought him thither. He complained of dyspnœa, which at first was not alarming, but afterwards became so severe that on the 1st July 1841, the day of his coming into the hospital, suffocation seemed imminent.

On admission, his countenance was anxious, pale, and somewhat œdematous: respiration was short, quick, painful, and panting. The patient kept in a half-sitting position. When he tried to turn on the left side, he felt an acute pain accompanied by great dyspnœa. The expectoration consisted of a thick yellowish mucus. There was found complete dulness on percussing over the whole of the sternum from its right margin to the anterior portion of the left side of the chest, from the second rib to the epigastric region, and laterally from the left margin of the sternum for six inches. Below the left clavicle, along the scapula, and in the axilla, the sound was clear; behind, on the same side, it was tympanitic. The whole of the front of the chest was clear on the right side to the sixth rib: from the fourth rib laterally, the sound was obscure. Behind, there was dulness increasing from above downwards: on the left side, there was tympanitic resonance. The liver was prominent and descended two finger-breadths into the hypochondrium. There was arching of the precordial region.

The impulse of the heart was imperceptible, and its sounds were very obscure. In the inferior sternal region, a friction sound was heard which it was difficult to distinguish amidst the noise of the mucous rattles. On the right side, below and in front, large mucous râles were audible, while behind, no respiratory sound could be heard.

The patient had some appetite for food; and had almost no thirst. The heat of the skin was normal. The pulse was small, irregular, and 112. There was neither diarrhœa nor constipation. The urine was of a deep red colour. The patient complained of a feeling of pressure over the epigastrium, and of pains in the precordial regions, when he leaned against the left side.

The diagnosis was:—extensive effusion into the pericardium, consequent upon an attack of pericarditis, compressing the lower part of the left lung, slight effusion into the right pleura, accompanied by infiltration of the pulmonary parenchyma, following pleuro-pneumonia and general bronchial catarrh.

Some relief was afforded by very active measures employed to promote absorption of the fluid: the pulse became less irregular. Percussion showed that the infiltration of the inferior lobe of the right lung was less, but that there was no diminution in the effusion into the pericardium. The patient was losing flesh. Mercurial preparations, from which most excellent results had been obtained, were uselessly pushed to larger doses, without producing diarrhœa or salivation.

On the 3rd August, three months after the date of this man's admission to hospital, incipient ascites was detected. It was then resolved to perform paracentesis of the pericardium; and on the following day, Dr. Heger operated. The place selected for the puncture was in the fifth intercostal space, and at a point about two inches from the left margin of the sternum, where the friction sound was not heard, and where there was less risk of wounding the internal mammary artery and large vessels. In the first instance, there flowed about twelve grammes of reddish serosity. Unsuccessful attempts were made to render the flow continuous by using in the first instance a catheter, and then a suction pump: a freer discharge, however, was obtained by getting the patient to hold in his breath and make an expulsive effort, strong pressure being made at the same time over the epigastrium. The fluid only came in jets under the influence of the systole. The serosity obtained was brownish red: at first, it was clear, and then it was flaky: in quantity, it was above 1500 grammes. During the operation the pulse was 112 and small. At intervals, the friction of the heart upon the canula was heard. Not one bubble of air penetrated into the pericardium. Almost immediately after the paracentesis, marked relief was experienced. The diaphragm regained its natural position, and the arching of the chest became less: the sound was clearer in the second intercostal space and along the outer margin of the scapula: the friction sound had disappeared. The extent of the dulness, however, led to the conclusion that there existed from seven hundred to eight hundred grammes of fluid within the pericardium. The wound was covered with a piece of diachylum plaster; and compresses soaked in iced

water were placed on the side, to prevent the reaction being excessive.

At 3 p.m., the patient had a shivering fit, and increased rapidity of breathing, but no cough. The pulse was 104. He passed a restless night, had some cough, and slight pain in the wound.

Next day, there was observed short rapid breathing, fits of coughing, mucous expectoration, a pulse of 112, constipation, dulness in the lower third of the left side of the chest, numerous subcrepitant râles, and a friction sound at the bottom of the sternum. With a view to check the pneumonia in the left lung, the patient was bled: the blood was very buffy.

Till August 10th, the pericardiac effusion went on increasing: the sound on percussion had again became dull in the second intercostal space: the friction sound was no longer audible: the sounds of the heart were more obscure: there was an increase in the intensity of the fever: the loss of flesh was becoming a greater cause of anxiety: *and it was feared that there was a development of tubercle.*

During the following week, there was an amelioration in the general state of the patient: the pneumonia of the left side was undergoing resolution, but there was effusion into the pleura of the same side.

On the 17th, there was diminution of the effusion on the right, and increase of the effusion on the left side. The pulse, which was small and irregular, ranged between 120 and 124. There was œdema of both malleoli and of the left leg. The dyspnœa was increasing and becoming complicated with excitement. Four days later, this œdema had become very great; and the local condition of the patient was the same as on his admission to hospital, but the cahexia was much more threatening.

On the 22nd, the pericardium was punctured: the result was the flow in drops of deep red fluid: every effort failed to render the flow continuous. The patient, with the canula *in situ*, lay on the edge of the bed for fully two hours, so that the serosity might be collected. In all, there was hardly obtained 400 grammes of a turbid bluish red fluid. For the canula, there was then substituted a caoutchouc tube firmly fixed, and closed at its free extremity by a moveable valve of pig's bladder. The liquid continued to flow through this tube from eleven in the forenoon to three in the afternoon; and the quantity may approximatively be estimated at 500 grammes. The

dulness continued after the puncture; but the movements and sounds of the heart became more distinct. The patient, exhausted by the long time occupied by the operation, experienced no relief from it. The pulse was 116. At five in the evening, the patient had a shivering fit. The tube was then removed, when there was again detected a pneumonia of the left side with bronchophony bellows-sound, râles, and characteristic sputa. Forty-eight hours later, this pneumonia passed into the stage of resolution; but the respiration still remained harsh.

On the 29th, there was a notable diminution in the amount of effusion; and there was a gradual progress in its absorption up to the 1st September. At that date, the pericardial friction-sound, which had returned, had ceased. The sound on percussion was almost normal up to the left nipple, and in the axilla: it was perfectly clear anteriorly in the first two intercostal spaces. The amount of œdema was insignificant: the cough was moderate, and the respiration was nearly natural: still, the patient continued to lose flesh.

On the 4th September, there were diarrhœa, œdema of the lower extremities and face, particularly on the left side. The sound on percussion on the upper and front part of the same side of the chest had again become tympanitic.

On the 11th, the left pleural effusion had made very great progress. There was complete dulness as high up as the axilla. Behind, there was enormous bronchial respiration; in front, respiration was harsh and whistling. It was impossible to measure with precision the extent of the pericardiac effusion. There was general anasarca up to within half an inch of the umbilicus, and there was ascites. The dyspnœa was extreme; the skin was cold and livid; asphyxia was becoming more and more threatening; and the pulse was too rapid to be counted. These symptoms increased in severity, and next day the patient sunk under them.

At the autopsy, the left lung was found free in the thoracic cavity, and the right was fixed by strong cellulo-fibrous adhesions. The left pleura contained from eight to nine pounds [pints], and the right five pounds of brownish serosity. The right lung, pushed back along the spinal column, was slightly compressed; its inferior lobe was dry and bluish, and the superior lobe was infiltrated with serosity which was partly frothy and partly unmixed with air. The left lung, also pushed back and compressed, had undergone similar changes, with

this exception, that there was a tuberculous cavity surrounded by a deposit of crude tubercle. The pericardium adhered to the ribs by its anterior surface from the second to the sixth rib. There were tuberculous glands in the anterior mediastinum. The pericardium was several lines in thickness; it adhered to the heart throughout the greater part of its anterior and posterior surface, and contained several ounces of yellowish flocculent fluid. By careful examination three layers of deposit on the pericardium could be distinguished; and the middle layer had undergone tuberculous degeneration. The heart was large and flaccid. Its dilated ventricles contained a black, soft coagulum. There was an ascitic effusion. The liver was of a brownish colour, and hypertrophied.

Gentlemen, the remarkable feature in this case is the pleural and pericardiac effusions progressing almost simultaneously: this happened also in the young man in whom I was obliged to perform in succession paracentesis of the chest and of the pericardium. The pericarditis developed itself slowly, almost without any acute stage, and without any violent inflammatory symptoms at the beginning of the attack. To make up for the absence of acute symptoms, there was extensive effusion, as in dropsy of the pericardium and in hydrothorax. Chronic œdema of the lung, anasarca limited at first to the lower extremities, and ascitic effusion, are almost necessary consequences of disturbance of the circulation; and therefore, in this case, we have no ground for being surprised either at their occurrence or duration.

I have intentionally dwelt upon the symptoms of tuberculisation existing during life, and upon the lesions characteristic of that condition being found after death. These, in fact, are the complications to which I formerly alluded, when I said that they were generally coincident with dropsy of the pericardium. You recollect, that in my lectures on hydrothorax, I pointed out to you that extensive pleuritic effusions of a chronic and latent character are frequently manifestations of the tubercuous diathesis, in this sense, that they affect individuals who, although they have not as yet any sign of tuberculisation, ultimately succumb to a tuberculous affection, which may or may not be an affection of the respiratory organs. In relation to this point I cited the case of the son of my colleague Dr. Thillaye, who died of tuberculous meningitis some months after having been successfully treated by paracentesis of the chest for extensive pleuritic effusion. Well then, gentlemen, it appears that a similar rule applies to dropsy of the pericardium. This, at least, was an

opinion expressed by my lamented colleague Dr. Aran. His personal experience, and attentive study of cases published by others, led my accomplished colleague to conclude that dropsy of the pericardium, when chronic and latent, generally coexists with tuberculous disease, and that these pericardiac effusions, which may be called symptomatic, are also generally those which assume such proportions as to necessitate paracentesis.

To conclude this historical review of paracentesis of the pericardium, let me remind you that Dr. Mérat mentions two successful cases of Dr. Remero of Barcelona;[1] and let me also quote from memory the practice of Dr. Bowditch of Boston, who, in desperate cases, has also performed this operation with success. I would also add, that in a discussion which arose in the *Société de Médecine des Hôpitaux de Paris* in relation to a case brought forward by Dr. Béhier, it was stated by Dr. Henri Roger that during his visit to Germany he had seen Professor Skoda puncture the pericardium without a successful result.

Gentlemen, Dr. Aran had twice occasion to perform this operation.

On a previous occasion, at the end of 1853 or beginning of 1854, prior to my operation upon the second patient to whom I referred at the commencement of this lecture, he attempted the operation, but had not the courage to complete it. After carrying an incision through the thoracic walls, and reaching the pericardium, he stopped short. When he felt the heart beating immediately under his finger, carried deep into the wound made by the bistoury, he was afraid to touch it, and so renounced the operation.

Who can blame the physician for showing such an excess of prudence under the circumstances? The operation had only in rare cases been brought to the test of experience; and the operations performed in France were but little encouraging. Notwithstanding the comparative certainty which has in our day been attained in the diagnosis of effusion into the pericardium, the diagnosis is still sufficiently difficult to leave room for the physician being deceived; and the special form of the dulness in pericarditis, very different from the rounded dulness due to hypertrophy of the heart, is a very uncertain sign. May it not be well to carry in the mind a case

[1] Dictionnaire des Sciences Médicales.

which, among many others, is well calculated to show that even with men the most able, errors are sometimes inevitable?

In 1841, or 1842, Dr. Vigla, now physician to the Hôtel-Dieu, when doing duty for Professor Rostan, found a young man in his wards suffering from dyspnœa approaching to asphyxia. He was unable to give any account of himself; and all that was known of him was that he had come out of the Hôpital du Midi. Over the precordial region, he bore the cicatrices of recent cupping. His general appearance and physiognomy indicated that he had recently had an illness.

On examining this young man as minutely as the circumstances permitted, Dr. Vigla found that the lungs were free from appreciable lesions; but that in the region of the heart, there was extensive dulness, with absence of normal or abnormal sounds; the pulse was very small and rapid. All who saw the patient concurred in diagnosing great effusion into the pericardium, recent in its origin, and the result of inflammation.

Certain death being imminent, prompt and decisive action was imperative. Under the circumstances, paracentesis seemed the only proceeding which fulfilled the indication; and Roux was asked to perform that operation. Roux proceeded with extreme caution, and made the opening by incision in preference to puncture: the result testified to his sagacity in so acting. When he reached the pericardium, and introduced his finger into the wound, he felt the heart beating, and recognised a slight friction between it and the pericardium, without, however, detecting the slightest degree of fluctuation. The operation was suspended, and inevitable death was expected.

The patient, without having inhaled ether or chloroform—the properties of which were not then known—was almost unconscious of what was done to him, and quietly sunk from asphyxia.

At the autopsy, there was found dilatation, which was quite a phenomenon [*dilatation phénomenale*] to use Dr. Vigla's expression, with attenuation of the walls of the heart: there were no valvular lesions, and no serosity in the pericardium.

The following case which occurred under your own observation corroborates still farther the point which I at present wish to establish:—A young woman, in November 1862, came into my wards in the Hôtel-Dieu and occupied bed 12 of St. Bernard's ward. She had been recently confined of her fourth child. She

complained of breathing with difficulty. The symptoms were dyspnœa, pale countenance, blue lips, anxious expression, œdema of the inferior extremities, and a small though regular pulse. The extent of the dulness in the precordial region, and the acute pain experienced when that region was percussed, testified to the existence of heart disease. This young woman had been suffering for a long time from palpitation of the heart, and the slightest exertion brought on difficulty of breathing. She stated that she had had several attacks af acute articular rheumatism. The cardiac affection was of a complex character. The great extent of the precordial dulness, the sounds of the heart seeming muffled, and as if distant, there being moreover a rasping bellows-murmur accompanying the first sound, heard at the base of the heart, and extending into the vessels of the neck, and the smallness of the pulse justified me in concluding that there was serous effusion into the pericardium, and contraction of the aortic orifice. Dr. Barth, who examined the patient at my request, concurred in my diagnosis: he also thought, that perhaps there were clots in the heart. In addition to the cardiac disease, we found the signs of general bronchitis, and slight effusion into the left pleura. Ought paracentesis of the pericardium to be performed? I hesitated. Next day, there was less oppression of the breathing, a diminution in the extent of the dulness, and less pain on percussion. After the lapse of some days, the pleuritic effusion was to a great extent absorbed, and by degrees the amelioration in the general state of the patient became so great that in opposition to my advice she left the hospital at the beginning of December, that is to say, in rather less than a month after admission.

After eight days' absence, however, she returned to the Hôtel-Dieu. Consequent upon slight fatigue, her difficulty of breathing had returned in an aggravated degree: her pulse was small and irregular, and although there was still great cardiac dulness, the bellows-murmur at the base, accompanying the first sound of the heart appeared more superficial: the dyspnœa soon increased, and the œdema of the inferior extremities also made progress: the pulse could not be felt in the radial arteries, and was hardly appreciable in the carotids: the extremities were cold. The oppression became greater and greater; and after a continuance for two days of symptoms of immediately impending death, the patient expired in a faint.

At the autopsy, the appearances found demonstrated that peri-

carditis had existed: false membranes of recent formation were found floating in a small quantity of serosity. There was great hypertrophy of the heart; and this was of itself sufficient to explain the extent of the precordial dulness. The aortic orifice was so contracted by calcareous deposits as hardly to allow the passage of a goose quill. The left pleura contained a small quantity of effusion, and some cellular false membranes. Both lungs were œdematous in their posterior and inferior parts: the bronchi were gorged with mucus, but the bronchial mucous membrane did not present any traces of recent inflammation.

The important point to remark in this case is that the effusion into the pericardium was never so great as had been supposed; for the extent of the precordial dulness nearly corresponded to the enormous hypertrophy of the heart. The feebleness and seemingly distant sound of the pulsations of the heart resulted from the feeble contractions of that organ, and not from a thick layer of serosity being interposed between the heart and the walls of the chest.

The two cases which I have just related prove then, that we cannot always affirm that the pericardium contains a large quantity of effusion, even when the majority of the signs of effusion are present; and consequently, it is always necessary to proceed with extreme caution in performing the manual operation, when it is supposed that paracentesis is indicated.

Dr. Aran's patient died from asphyxia, occasioned by dropsy of the pericardium. On opening the body after death, there was found the effusion diagnosed during life—effusion sufficient in quantity to obviate the fear of wounding the heart when making the opening into the pericardium; and the only concomitant lesions were some tuberculous granulations on the pleura. The operation evidently offered great chances of success in such a case; and Aran was resolved not to let the opportunity slip should a similar case present itself. He did not require to wait long.

On the 6th November 1855, my lamented colleague read to the Academy of Medicine "a case of pericarditis with effusion treated successfully by tapping and iodinous injections." I shall now read this case to you in the form in which it was published at the time.[1]

"The patient was a smelter of metals, a young man of twenty-three or twenty-four years of age. Though of a miserably delicate con-

[1] Bulletin de l'Académie de Médecine, t. xxi, p. 142.

stitution, he had never suffered from serious illness, except when he was for a month in my wards, at the close of 1854, for a pleurisy of the left side with extensive effusion. He left the Hôpital Saint-Antoine in fair health on the 21st of last November. A month afterwards, he perceived a pain in the chest, near the third or fourth rib, accompanied, when at his work, by a little difficulty of breathing and some palpitation of the heart. The pain continued till the fine season set in, but with the warm weather it disappeared. This young man was consequently in very good health, when towards the middle of last July, he was seized with fever, cephalalgia, shivering, pains in the back, decided pain under the left nipple, palpitation, and dyspnœa.

"On the 27th July, the date at which he came into my wards, there could be no doubt of the existence of pericarditis with extensive effusion. On the one hand, the patient had high fever, marked by intense heat of skin, cephalalgia, urgent thirst, and a pulse of 116: on the other hand, the local signs were more characteristic—there were lancinating pains in the fourth and fifth intercostal spaces on the left side, in front, which were increased by pressure: there was also very great sensitiveness over the epigastrium, when pressure was made with the hand: there was greatly increased precordial dulness commencing superiorly below the third rib, and extending inwards to the right synchondrosternal line, measuring 12 centimeters vertically, and 14 centimeters transversely: the impulse of the heart was almost imperceptible, and its sounds seemed as if muffled and distant.

"The wretched constitution of this patient, and probably old date of the beginning of the heart disease did not encourage me to subject him to a very energetic antiphlogistic treatment. Moreover, for eight days he had had looseness of the bowels, and a state of chest somewhat doubtful, there being on the left side in particular, diffused sibilant râles: this condition still less induced me to resort to large bleedings. On the first day, therefore, I took blood by six cupping glasses, administered calomel internally in small doses; and with a view to induce rapid salivation, I ordered the front of the chest to be rubbed three times a day with mercurial ointment.

"This treatment was not in any degree successful. I tried in vain to assist it by applying in succession two large flying blisters to the precordial region. Not only were the symptoms not arrested, but the effusion increased day by day, and with this, the impediment to

respiration and circulation became augmented. Before three days had elapsed, the pulse had become feeble, irregular, unequal, and very rapid. I persevered in the administration of mercurials, but had great difficulty in even slightly affecting the gums.

"As the character of the pulse was becoming more and more indicative of an impending aggravation of the symptoms, I soon found that it was imperative to take action. On the 7th of August last, at the morning visit, I was told that the previous day and night had been frightful: the patient had been almost suffocated, and had been obliged to pass the night sitting up in bed. The respirations were 40, and the pulse 120 in the minute: the pulse was irregular, intermittent, and unequal. The embarrassment of respiration and circulation were only too well explained by the local signs: there was dulness extending two centimeters beyond the right edge of the sternum, and measuring transversely from 14 to 16 centimeters, and vertically 12 centimeters: below this situation, the sounds of the heart were inaudible; there was an absence of impulse: the liver was pushed downwards and to the left of the median line.

"What ought to be done? Ought the same treatment to be continued. Its inutility was certain. Ought antiphlogistic measures to be resorted to? The weak condition of the patient explicitly contraindicated such means; and moreover, it was necessary to relieve the patient at once, or leave him to die within a few hours. I resolved to tap the pericardium."

Dr. Aran selected the same mode of operating which was adopted by Professor Jobert in the case in my wards; that is to say, penetrating with a trocar through the fourth or fifth intercostal space direct into the cavity of the pericardium.

"However," in continuation, says Dr. Aran, "I was not without uneasiness as to the result of using an ordinary trocar; and so, with a view to avoid a possible mishap, I employed a capillary trocar, such as I have successfully operated with in hydatid cysts of the liver. Thus, I found myself more at my case, being convinced that a puncture of the heart by a capillary trocar could not be followed by mortal hæmorrhage into the pericardium. But was there no way of absolutely guarding against this accident? This security I expected to obtain, by adopting the following precautions.

"The circumference of the pericardium was circumscribed by a series of concentric lines of percussion proceeding towards the heart from different parts of the chest, and the shape of the surface of the

dull part being thus carefully delineated, I endeavoured, by the ear, to limit the zone within which the sounds of the heart were quite inaudible, that in which they were slightly heard, and that in which they were distinct. The sounds of the heart could not be heard in the lower part of the dull region, and seemed as if muffled and remote in the fourth intercostal space, to the inside of the nipple: they were absent in a space of sufficient extent to enable the operator if necessary to insert the trocar from before backwards without risk of wounding the heart. For greater safety, I selected a point in the fifth intercostal space, at which I made an incision in the skin with the lancet, and then, slowly introducing the trocar from without inwards, and somewhat from below upwards, after having once withdrawn the inner stylet without seeing any flow, I reached the pericardium by two stages: there immediately occurred a spurting gush of fluid, affording satisfactory proof that the sac had been penetrated.

"The tapping of the pericardium was certainly accomplished within a shorter space of time than I have taken to describe the operation. Those only who can recollect the feelings with which they performed this operation for the first time, can estimate my anxiety at the commencement of the proceeding, and my lively satisfaction, my extreme comfort, when I saw the evacuation of the fluid progressing. I withdrew by the trocar about 350 grammes of reddish transparent serosity. At first, it came in spurts, and afterwards it dribbled out; but the patient aided the flow by efforts which he prolonged as much as possible, in consequence of the decided relief which he experienced from the evacuation of the fluid.

"The sounds on percussion corresponded with the progressive diminution of the dulness arising from the evacuation of the fluid; and on auscultation, they were heard, more and more distinctly, unaccompanied by a rubbing sound. The pulse became fuller, more regular, and less frequent: it fell from 120 to 96 in the minute.

"I might have rested satisfied with having performed a single palliative tapping; but I thought I could do something more for the patient. Relying upon the success I had obtained in pleurisy, I slowly injected an iodinous solution composed of 50 grammes of water, 15 grammes of tincture of iodine, and one gramme of iodide of potassium. I did not feel very comfortable in respect of this injection. I asked myself:—what is going to happen? I said has it not been alleged that the pericardium is endowed with excessive

sensibility? The injection, however, was not even felt. After having retained the injection in the pericardium for some moments, I allowed some grammes of it to escape, and then closed the wound by means of graduated compresses, and by placing a bandage round the body.

"The results of this tapping were very simple, but the fluid was reproduced, and ere long the patient lost a great part of the ground which he had gained by the operation. Respiration became more embarrassed, and the pulse irregular and more rapid. The dulness, which had at first seemed to diminish, increased, particularly in a lateral direction. There was manifest arching of the chest. The pulsations of the heart were deep-seated. To be brief:—on the 19th August, twelve days after the first tapping, I tapped a second time; and, as before, between the fifth intercostal space, following, moreover, exactly the same proceedings as on the previous occasion. At this second operation, I evacuated 1,350 grammes of a very albuminous greenish liquid, in colour resembling bile. As on the first occasion, this fluid came in gushing spurts, but it afterwards dribbled out. The patient, feeling relief from the flow of the fluid assisted me by his efforts which it was necessary to restrain through fear of allowing air to penetrate into the pericardium: however, air did penetrate after the injection of the solution of iodine, the strength of which was now increased to fifty parts of tincture of iodine to the same quantity of distilled water, with the addition of four grammes of iodide of potassium: nearly the whole was allowed to flow out again. I was consequently enabled to detect the curious sign of hydropericarditis, for the description of which we are indebted to Dr. Bricheteau, viz. an excessive gurgling, a sort of churning sound [*clapotement*] like that produced by a pump jumbling air and water together in the same cavity. After the operation, the precordial region was the seat of well-marked tympanitic resonance.

"The results of the second were not less simple than the results of the first tapping; but the relief experienced was still more marked, for some hours after the operation, the gurgling and tympanitic resonance had disappeared from the pericardium. On the very evening, however, of the day of the operation, the reproduction of the effusion began. Up to August 21st, the extent of the dulness seemed to be increasing: on the 22nd, it remained stationary: and from the 23rd, it began to diminish, particularly below and laterally. The sounds, though feeble, soon began to be perceptible at the apex

of the heart; and from August 28th, the dulness did not extend inwards beyond the median line, nor outwards beyond the nipple, nor superiorly beyond the third rib.

Notwithstanding this apparently favourable progress of the disease, the young man still had other dangers to incur. The chest affection under which he was suffering at the date of his admission to hospital, was not remaining stationary; and in proportion to the degree in which the heart symptoms seemed to moderate, the signs of pulmonary tuberculisation became more and more evident, particularly in the left lung, in which, at first, I had noted symptoms of inflammation. This was not all: towards the end of September, the ankles became œdematous, and some days later, the swelling invaded the scrotum, the lower extremities, as well as the thoracic and abdominal parietes.

"Since the end of October, the œdema has been completely gone —thanks to his youth, and thanks also, probably, to the application of numerous flying blisters to the chest, and the use of vapour baths. There has seemed also to be a gradual amelioration in the thoracic phenomena: with return of appetite, there came return of strength: respiration regained its freedom, and with the exception of a continuance of cough at night the patient may consider himself as completely cured of an affection which had brought him to the very brink of the tomb. Need I add, that the physical signs of pulmonary tuberculisation still remain, notwithstanding the amendment which has taken place in the general and local condition of the patient?"

I do not feel at all afraid, Gentlemen, of having trespassed on your patience by reporting at length this case so full of interest from every point of view: it is not, moreover, the only case of tapping the pericardium which Aran had to record: a short time before his death, he told me that he had thrice performed, and thrice performed successfully this operation.

These cases, other cases which have occurred in my own practice, and cases which might now probably be added, conclusively demonstrate that paracentesis of the pericardium is not beset with the perils which for so long time frightened experimenters, but existed only in their own imaginations.

Were it not for the diagnostic difficulties presented by dropsy of the pericardium, difficulties very much more serious than those which sometimes occur in the diagnosis of hydrothorax, tapping the

pericardium would be as simple an operation as paracentesis of the chest or abdomen. Indeed tapping the pleura, though in most cases, exempt from danger, is more calculated than tapping the pericardium to excite fears, and lead to bad consequences.

Not only is paracentesis of the pericardium free from risks; but experience appears likewise to have fully established the safety of using injections for the radical cure of dropsy of the pericardium. Thus, therefore, are realised the anticipations of Richerand who was the first by whom the idea was conceived of applying to effusions into the pericardium that treatment which is employed every day in cases of hydrothorax, ascites, and effusions into joints. Professor Bouillaud in his "*Traité Clinique des Maladies du Cœur,*" without venturing to give a decisive verdict on paracentesis of the chest and the value of iodinised injections, enunciated the following opinion in the second edition published in 1841:—"an exaggerated notion has probably been entertained of the dangers of pericarditis, a condition which must be produced before it is possible to have adhesion of the opposite surfaces of the pericardium, the sole means of preventing a reaccumulation of the fluid evacuated by the tapping:" and he adds that "pericarditis produced by irritant injections would be a proceeding of the simplest possible description."

Gentlemen, I have now a few words to say on *the operation* itself.

Several points have been proposed as the most suitable for opening into the pericardium. As I have already told you, Senac, Skielderup, and Laennec recommended that the sternum should be trepanned immediately above the ensiform cartilage, and, with a view to fix the spot with more precision, they advised the puncture to be made below the insertion of the cartilage of the fifth rib.

Larrey believed that it was easier and more convenient to make the puncture between the edge of the ensiform cartilage and the cartilage of the eighth rib on the left side. By carrying the instrument from below upwards, and a little to the right, the pericardium will be reached he thinks, with greater safety, and in such a way as to give freer egress to the fluid. I at first looked on this method as very rational: but on recollecting that surgeons have properly called attention to the risk of encountering a branch of the internal mammary artery which is sometimes of such a calibre as to reach to the ensiform cartilage; and reflecting also on the fact that—in accordance with a remark of Professor Velpeau—the instrument

might be carried in such a direction as to avoid the pericardium in subjects whose œdematous state or plump condition was sufficiently decided to prevent the skin from coming into immediate contact with the cartilage and ensiform appendix, I renounced that method.

The place which I select as the most favourable for the operation is that which M. Jobert and I chose in our own two patients—the fourth and fifth intercostal spaces. The precautions adopted by Aran, and pointed out in his case are useful: when an occasion occurs, you will do well to put his precepts into practice.

Tapping may be performed either directly by means of the trocar, or by incising with a bistoury, layer by layer, the thoracic walls and the pericardium, or by adopting a mixed proceeding; that is to say, by first cutting through the superficial layers, and then puncturing the subjacent tissues by the trocar.

I admit that there is something in the simplicity of the proceeding which renders it a more attractive operation to puncture with the trocar than to cut with the bistoury. But let me repeat, that the diagnosis of dropsy of the pericardium is not always so easy as is alleged: in making a direct puncture with the trocar, I should be afraid of coming upon the heart. This is a risk which I should dread even more in a case in which there really was effusion into the pericardium, for the heart, in place of flying before the instrument, might as has been well expressed by Senac, come up to meet it, and thereby be run through. Even when using Aran's capillary trocar, I should still be very far from being without anxiety on that score.

The only inconvenience which I can see in using the bistoury, is that some of the fluid effused into the pericardium may fall into the pleura, flowing out between the edges of too large an incision, and the canula introduced into the pericardium. No importance attaches to this inconvenience: in fact, it has been shown by experiments made on wounds of the chest—a subject which I discussed with you at some length the other day—that blood effused into the pleural cavity is very rapidly absorbed; and the same rapidity of absorption ought to take place in respect of a serous effusion.

There is, therefore, no danger in allowing a fluid which is even less irritating than blood to fall into the pleura. Mark well this fact:—if the fluid effused in a case of pleurisy is not absorbed, it is either because the pleura is still in a state of inflammation, or because its surface is coated with false membrane, and consequently in a condition

unfavorable to the accomplishment of absorption. When there is no morbid condition of the pleura, absorption goes on well, the symptoms are of a less serious character, and recovery is more likely to occur.

If I were called upon to-day to puncture the pericardium, I should modify the operation in the spirit of the remarks I have now made. I should make my incision through the skin immediately external to the sternum, about the fifth, sixth, or seventh sterno-costal cartilage, selecting, as Aran recommends, the point where the dulness is greatest, and where it is most difficult to perceive the movements of the heart. I should try to penetrate between two cartilages, keeping as near as possible to the sternum. At the sternum, the cartilages touch one another; but by employing a spatula, or any such like suitable lever, I should be enabled somewhat to separate the edges of the two cartilages, and, if it were necessary, I should not hesitate to remove as much cartilage as would allow the pulp of the finger to reach the pericardium. The case reported by Dr. Vigla shows how necessary it is to be assured by digital examination that there is a sufficient distance between the heart and the point at which the pericardium is punctured.

To facilitate the escape of the fluid, the most important circumstance to attend to is to allow the canula of the trocar to remain some time in the pericardium; and it is useless to practise the different manipulations which have been recommended as calculated to accelerate the evacuation. Suction pumps afford no assistance, and give a troublesome complication to the operative apparatus. However, the membranous valve which I employ in paracentesis of the thorax for pleuritic effusions may, without producing any inconvenience, be attached to the free extremity of the canula, although its utility is an open question.

As soon as the canula has been introduced into the pericardium, the liquid begins gradually to flow out. Aran observed in his cases, that the continuous jet which the fluid formed was sometimes projected to a great distance in spurts during deep inspirations—a phenomenon which he attributes to the pressure of the lung on the pericardium. This phenomenon did not present itself in my two patients, nor was it noted in the cases which I have reported to you.

When the canula is removed, all that is necessary is to close the wound with a diachylon plaster kept in its place by a bandage round

the body. The wound requires no treatment: it hardly occasions any pain, gives rise to no great amount of inflammation, and never leads to suppuration even of very limited extent.

Gentlemen, paracentesis of the pericardium is decidedly indicated only in cases in which life is threatened by the extent of the effusion. The occasions on which it ought to be resorted to must always be of rare occurrence.

Simple idiopathic dropsy of the pericardium uncomplicated with any other dropsy, or with any serious lesion of the thoracic organs is certainly seldom met with. Generally, profuse effusion into the pericardium is only one of the manifestations of a state of disease which is not exclusively localised in that situation, but also attacks other essential parts of the economy.

I have told you that Aran enunciated an opinion to the effect that effusion into the pericardium sufficiently profuse to necessitate paracentesis, is generally coincident with the tubercuIous diathesis. I have informed you that this opinion was based on facts observed by our lamented colleague. His own two cases, and those by the recital of which I began this lecture, completely support this view.

But if we cannot hope in such cases, to cure the patient by withdrawing the fluid from the pericardium, we are at least certain of relieving suffering and prolonging life by removing a serious complication involving imminent danger. Had paracentesis of the pericardium no other reliable claim, this would entitle it to a place among operations worthy of being retained and sanctioned. When we witness the anxiety produced by the pressure of fluid on the heart, when we witness the fearful and protracted agony resulting from such a state, we are only too happy to have it in our power to afford even temporary relief, and to be able to prolong a life which we have rendered less painful to endure.

LECTURE XXXVIII.

ORGANIC AFFECTIONS OF THE HEART.

General Considerations.—Insufficiency of the Aortic Valves is the most serious of all the Lesions of the Cardiac Orifices.—Dropsy treated by Purgatives.—Diarrhœa sometimes requires to be arrested: at other times it constitutes a natural crisis which ought not to be interfered with.—Diagnosis of Affections of the Heart is often difficult.—Embolism and its Consequences.

Gentlemen:—A woman who, on several occasions, has been a patient in our clinical wards, will afford me an opportunity of presenting some general considerations, pathological and therapeutical, in relation to some peculiar symptoms which arise in the course of organic affections of the heart—considerations which you will be able from time to time to apply at the bedsides of our patients.

The woman to whom I refer laterally occupied bed 34 of St. Bernard's ward: she came into hospital on account of complications dependent upon an affection of the heart of very easy diagnosis. Upon auscultation, the cardiac lesion was revealed by a double bellows-murmur, having its maximum intensity in the situation of the apex of the heart: the blowing accompanying the first sound of the heart was harsh, and that accompanying the second was softer. These stethoscopic phenomena were characteristic signs of valvular insufficiency, and of contraction of the left auriculo-ventricular orifice.

My present object is not so much to call your attention to organic lesions of the heart, as to show you the very great difficulty of making a confident prognosis; and to point out to you certain rules for the treatment of some of the complications which arise consequent upon the lesions.

I would remark, however, Gentlemen, in respect of valvular insufficiency, that it is generally coincident with contraction of the orifice. In fact, the causes which most commonly prevent the valves from fitting closely to one another are changes of these membranous partitions. Their thickening, their induration, their fibro-cartilaginous, osseous, or petrous transformation, their partial destruction at their free margin, their perforation, their more or less extensive rupture at the base or centre, the presence of vegetations on their surface or edges, and all kinds of structural change, which, coexisting with more or less considerable thickening and induration of the valves, prevent them from performing their functions in a perfect manner. This thickening and hardening, and the presence of somewhat bulky vegetations at the edges or upon the surface of the valves, irrespective of valvular insufficiency, necessarily narrow the orifices at the entrance of which the valves are placed.

This valvular insufficiency and contraction of the opening give rise to more or less impediment to the circulation of the blood in the heart, and this again produces a series of phenomena, some of which are local and pertain to the heart, while others are general and belong to other organs.

Some of the local phenomena are subjects of complaint by the patients. Among these are palpitation, a sense of embarrassment and weight in the precordial region or towards the pit of the stomach, which augment the muscular effort when it is necessary to exert a little more than usual, as, for example, in going up a stair. At a more advanced stage of the disease, there is greater or less difficulty of breathing. These symptoms, however, are often absent. Other local phenomena, the existence of which is made out by exploration of the heart in different ways, afford us more certain diagnostic signs of the lesion.

When the disease has somewhat advanced, simple inspection of the precordial region gives us some information as to the disturbance of the heart's action, and application of the hand enables us still better to appreciate it. In fact, on applying the hand, we can recognise that sort of undulatory movement, that vibratory thrill, called the purring fremitus [*frémissement cataire*] which is coincident with irregularities, intermittencies, and inequalities of the pulsations of the heart. Auscultation by the ear, or by the aid of a stethoscope, furnish signs consisting of bellows-murmurs of great diversity, the physiological explanation of which has been, and still

is explained by different theories, which I do not think it necessary to discuss here in detail. Let me say, however, that the beautiful experiments on horses performed by M. Chauveau no longer leave any room for doubt as to the cause of the normal and abnormal sounds of the heart: to those present at the experiments of which I speak, it was clearly demonstrated that Rouannet's theory is that which is alone admissible.

Percussion enables us to recognise increase in the volume of the heart, whether that increase be dependent upon dilatation of the cavities or hypertrophy of their walls. These alterations of the heart, almost invariably coincident with lesions of the orifices, are the necessary consequences of impediment to the circulation of the blood. The mechanism of their production is easily understood. From the moment that the muscular contractions of the heart are inadequate to overcome the obstacle to the passage of the blood out of the cavity which contains it, the walls of that cavity gradually become distended by the accumulation of blood, and in this way the cavity itself is dilated. This dilatation is seldom simple, that is to say, only the result of attenuation of the parietes: generally—almost always—the dilatation is accompanied by hypertrophy of the walls of the heart, originating chiefly in an excess of muscular action. Though I do not wish at present to discuss a question of general pathology, I would nevertheless call on you to observe, that there takes place in the heart a change similar to that which occurs in other hollow organs in which we see dilatation along with greater development of the muscular fibres from the existence of an obstacle to the exit of the contents requiring increased efforts for the accomplishment of the normal expulsatory function. The same takes place in the heart, as in the bladder, the bronchial tubes, the stomach and other portions of the digestive canal. This hypertrophy of the heart, as has been justly remarked by clinical observers, by Hunter, Laennec, Beau and others, is a lesion specially and providentially employed by nature, as a means of overcoming the obstacle to the circulation of the blood. The result is, the maintenance for a certain time of the performance of a function essential to life. I have stated that this lesion is to a great extent produced by an excess of muscular action; but while I say so, I admit, that we must also take into account as a cause of the hypertrophy, the pathological change in the muscular tissue brought about by that morbid action conventionally termed inflammation or irritation.

Let us now return to the consideration of embarrassment, more or less considerable, to the circulation of the blood in the cardiac cavities. Gentlemen, if I do not pause to study with you the precise diagnosis of the seat of the lesions of the orifices, it is because—as has been admitted by an eminent physician, whose competence to express an opinion on such a point no one will gainsay—the study is essentially one more curious than useful.[1] Nevertheless, the differential diagnosis of insufficiency of the aortic valves is of very great importance in practice. The most frequent causes of sudden death are those which depend upon lesions of the aortic valves; and it is likewise a fact recognised by most practitioners that these are the very lesions least frequently accompanied by that assemblage of morbid phenomena which constitute the general symptoms of diseases of the heart.

Let us see what are these *general phenomena*.

Disturbance of the functions of the heart must necessarily produce decided effects throughout the whole circulatory apparatus. Appreciable modifications of the arterial pulse, of the state of the veins and capillaries, show that the circulation is embarrassed. The pulse, irregular, unequal, and intermittent, as are the pulsations of the heart, is generally small; but when there is considerable hypertrophy of the left ventricle, the pulse is also hard and vibrating, presenting sometimes, a peculiar fremitus, which is most distinct in the carotid, subclavian and radial arteries. In some cases, the arterial pulsations succeed one another stroke by stroke, and this reduplication of the pulse is coincident with a regurgitant murmur heard on auscultating the heart. The insufficiency of the aortic valves is characterised by a bellows-murmur at the base, accompanying the second sound of the heart, and by a bounding pulse with flexuosity of the radial artery: this last-named sign originally pointed out by Selle, and very specially insisted upon by Corrigan, is of great diagnostic value in this affection.

Marcy's *sphigmograph* gives a good representation of the peculiar

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[1] Professor Bouillaud, in his "*Traité Clinique des Maladies du Cœur*" (2nd edition, vol. ii, p. 362), says:—'Do distinctive signs exist by which we can ascertain the precise seat of contraction in one or other of the cavities of the heart? The solution of this problem, which is essentially more curious than useful (*qui est au fond plus curieux qu'utile*), shall now engage our attention for a few minutes."

characteristics of the pulse in *aortic valvular insufficiency*. You are aware that this ingenious instrument, by means of a pen attached to the arm of a lever resting by one end on an artery, each pulsation of which raises it up, delineates the arterial pulsations upon a strip of paper which goes on unrolling. Well then! Corrigan's special bounding of the pulse, which strikes the finger smartly like a trigger, is expressed on the slip of paper of the sphigmograph by an ascending vertical line terminating in a sharp point or in a sort of hook, after which comes an oblique descending line more or less flexuous in the middle. The entire tracing consists in a series of vertical and oblique lines joined by the point or hook. The height of the vertical line is in proportion to the force of the arterial diastole.

But in the pulse of *contraction of the aortic* orifice, the ascending line of the tracing, corresponding to the arterial diastole, is not vertical, but oblique; and the descending line is oblique in an inverse direction, and flexuous. The ascending line never attains that height which it reaches in the pulse of aortic valvular insufficiency.

In insufficiency of the mitral valve, the pulse is almost always irregular, and of an irregularity which is absolute, and in no respect typical; that is to say, it is not represented in any uniform manner upon the sphigmographic tracing. The pulse has less volume: and so feeble are some beats that it is almost impossible to appreciate them by the finger. On the tracing, the arterial diastole is figured by vertical lines of unequal height, and the systole by oblique tremulous lines of the most irregular form.

In *contraction of the mitral orifice*, the pulse is regular, and the sphigmographic tracing greatly approximates to the normal. In the cases in which there is a presystolic bellows-murmur, the nature of the lesion may be diagnosed by the negative characters of the pulse.[1]

When disease of the heart has reached a pretty advanced stage, the existing impediment to the venous circulation is indicated by swelling of the veins near the heart—those for example of the neck and face; and this turgescence is particularly obvious in the external jugular veins, where it is sometimes accompanied by undulatory pulsations, analogous to and synchronous with the arterial pulse. This is the "*venous pulse*," which Lancisi (who seems to have been the first to observe) gave as the sign of hypertrophy of the right

[1] See MAREY's work entitled:—"Physiologie Médicale de la Circulation du Sang." Paris, 1863.

ventricle. It is caused by reflux into the veins of a certain portion of the sanguineous tide which the right auricle has been unable to send into the ventricle: it is thus caused, whether there be contraction of the auriculo-ventricular orifice, whether there be insufficiency of the tricuspid valve permitting the blood in the ventricle partly to regurgitate into the auricle, or finally, whether, by reason of the obstacles which the blood encounters in passing from the right into the left cavities, the ventricle is unable to empty itself completely.

In addition to the embarrassment of the venous, there is embarrassment of the capillary circulation, which declares itself by a livid tint of the skin, swelling of the face, puffiness of the eyelids, a bluish colour of the lips, and more or less injection of the skin of the extremities.

The morbid functional phenomena which occur are dependent upon disturbance in the capillary circulation. The *embarrassed respiration*, at first consisting in some breathlessness after rather violent exercise, such as walking more rapidly than usual, increases in proportion as the affection of the heart makes progress, and at last reaches a high degree of dyspnœa: the *disturbance of the cerebral functions*, which supervenes in the last stage of the disease—the *sanguineous congestion* which occurs in the principal viscera, the lungs, liver, spleen, and encephalon, and which sometimes proceeds to the extent of *hemorrhage* (pneumohemorrhage for example, a frequent complication of heart diseases), or induces structural changes such as *cirrhosis* of the liver [1]—and finally, dropsical affections such as œdema of the extremities, anasarca, effusion into the serous cavities:—all these phenomena are *chiefly* consequences of mechanical obstruction of the circulation.

I say *chiefly* the result of mechanical obstruction, because mechanical obstruction is not sufficient of itself to explain the production of the morbid phenomena of which I am now speaking. So true is this, that we sometimes see individuals rapidly succumb after having presented all the general and rational symptoms of cardiac disease, and yet in whom it was never possible during life to recognise well marked local signs of such an affection, and in the *post mortem* examination of whose bodies there were not found any lesions of the heart sufficient to explain either the symptoms observed or the death; and we likewise see persons presenting all the physical signs of a

[1] See a subsequent lecture on CIRRHOSIS.

disease of the heart live for a long time without appearing to experience any notable derangement in their health.

If we only take into account the anatomical lesion, an organic affection of the heart is in reality not a disease. Should any one be astonished at this assertion, let me ask him if he would regard as a disease, slow progressive asphyxia induced by passing a noose round a man's neck and daily tightening the cord to so slight an extent that it would take two years to cause death? Organic affections of the heart, however, are always, or nearly always, more than a simple mechanical obstruction to the central circulation: the localised morbid affection, which has occasioned the formation of the material obstacles, is also to a great extent the cause of all the organic and functional disorders which arise.

This, truly the most medical point of view from which to consider diseases of the heart, is that adopted by many clinical physicians: it has recently been admirably expressed by Dr. Mauriac in his excellent thesis, from which, with your permission, I shall read some passages.[1]

"When," says Dr. Mauriac, "we have to estimate in a general manner the causes of death in persons affected with diseases of the heart, it is indispensable, if one wishes fully to grasp the problem, and to look at the question in a manner at once philosophical and medical, to examine in the first place the share which certain diatheses have in the production of the secondary phenomena of these diseases, when, after a period of longer or shorter duration, they throw the economy into a peculiar state of cachexia, which is conventionally designated *cardiac cachexia*. It is known that this special cachexia is the source of profound modifications in the crasis of the humours; and that the two principal phenomena are an asthenic condition of the circulation in all the splanchnic viscera, whence result passive congestions of these organs, and an abnormal exhalation of serosity into the cellular tissue and serous cavities. These signs of general disturbance of the system show themselves sometimes at so early a period, at a period so close to the first manifestation of the local symptoms of heart disease, that it becomes a question whether the disease, regarded as a whole, is primarily local or general. Where does it begin? Is its origin in the heart alone?

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[1] Mauriac:—Essai sur Les Maladies du Cœur.—De la Mort Subite dans l'Insuffisance des Valvules Sigmoïdes de l'Aorte. [Thèse de Paris, 1860.]

Is it from the heart that there comes that morbid impulse the evolution of which will soon involve the entire economy? Or, must we seek for its origin in all parts of the circulatory system? Or again, is the entire circulatory apparatus simultaneously affected; and is it not the heart which is chiefly affected, because in its central action is comprised, so to speak, all the forces which put in motion that fluid which pervades and nourishes all our tissues? These are great questions in general pathology to which it is not easy to reply.

"It is now an accepted scientific fact, verified by every day's experience, and placed beyond dispute by the beautiful researches of Dr. Bouillaud that there is a primary diathesic cause for nearly all diseases of the heart. It matters little, whether this general morbid state, which concentrates its energy on the organs of circulation is purely inflammatory or essentially rheumatic or gouty; but it is important to remember that every diathesis causes every molecule to live a life specially morbid, and consequently exercises upon the entire economy a profoundly debilitating influence. If every diathesis weakens the force of the organism by modifying the physiological performance of elementary nutrition, may not the various diatheses which originate diseases of the heart do this much more certainly by attacking the apparatus which conveys to all parts of the body the fluid whence assimilation derives the materials by which the tissues are nourished? Herein lies, does it not, the primary cause of that general morbid deterioration of which we have to take account?

"This is not all: the pathological processes peculiar to each diathesis, and from which it derives its characteristic physiognomy, disorganise the cardiac tissue, and so begins a series of secondary phenomena, which have been too exclusively ascribed to the existence of impediments to the circulation of the blood in the cavities of the heart. Some pathologists have even gone farther than this in localising the causes of the morbid phenomena; they have ascribed these impediments to the circulation solely to the material obstacles situated at the orifices. The lesions of the orifices—contractions or inadequacies—only express one of the phases of the pathological changes of which the heart is the theatre: the danger which they occasion is only relative."

Dr. Mauriac supports these propositions by cases, such as I have just been pointing out to you, of persons who, though they have the

physical signs of apparently serious disease of the heart, live for a very long time, and moreover without presenting the symptoms of general disturbance of the economy apparently imminent from the certainty that there existed an obstacle to the passage of the blood through the heart. He then thus continues:—

"The doctrine regarding diseases of the heart which is exclusively based upon considerations referring to material obstacles to the passage of the blood does not solve every difficulty nor remove every doubt. The attractiveness of the doctrine arises from its simplifying the phenomena by making them subordinate to a mechanical cause which the mind can appreciate much better than a vital or diathesic cause. But the operations of nature are essentially complex: a pathological phenomenon which at first sight seems to be simplicity itself, implies a change, permanent or temporary, in so many elements, or an exaltation of so many organic functions, that there is a great risk of only seeing one side of the truth, and leaving the others in the shade, if we found a theory solely on one class of phenomena."

In reality, a practical fact is dominant in the pathology of the heart: the diseases of this organ are those which most deceive the physician at the bedside of the patient: their diagnosis is simple, but it is quite otherwise in respect of their prognosis. The course of the disease, and its possible complications, are subordinate to very many circumstances, some of which—such as the intercurrent affections—are recognisable, but the majority of which elude recognition.

Speaking in a general way, it may be said, that a predisposition to pulmonary affections, that an exaggerated susceptibility of the nervous system, place those suffering from diseases of the heart in an untoward position in consequence of the manner in which pulmonary and nervous affections react on the central organ of the circulation. The former tend to occasion stasis of the blood in the right side of the heart, and consecutively in the entire venous and capillary system, thereby leading to passive congestions and serous effusions: the second are direct causes of functional disorders which greatly complicate the organic disease. But leaving generalities, I ask:—who can explain why a particular individual, with the exception of the morbid conditions of which I have been speaking, may go on for a long time without experiencing much derangement of health notwithstanding an extensive cardiac lesion, while another

person sinks rapidly under an organic disease of the heart, the local symptoms of which were much less serious, their seat in other respects being the same? I make this latter distinction, because, as I have already said, the insufficiency of the sigmoid valves of the aorta is of all cardiac organic affections the most serious, as well as one of the most frequent causes of sudden death, although it is the least frequently attended by general symptoms during life.

Gentlemen, let us now resume consideration of the cases at present under our observation: let us return to the patient occupying bed 34 in St. Bernard's ward.

This woman was admitted four or five months ago, suffering from extensive anasarca. Besides infiltration of the cellular tissue of the lower extremities, and puffiness of the face, she had pulmonary œdema, which, on auscultation, was found to be characterised by sibilant and subcrepitant râles, heard principally at the base of the lung. So greatly was respiration embarrassed, that death from asphyxia seemed likely to occur within forty-eight hours. The affection of the heart, however, judging from the analysis of the symptoms which I was enabled to make (though with difficulty in consequence of the greatly embarrassed state of the respiration and circulation) was not more serious than it is to-day. It was not the first occasion on which the patient had had symptoms similar to those which I was then called upon to relieve. Under the circumstances, all that I could do was to attack the general dropsy, under the impression that by promoting the evacuation of the infiltrated fluid, by freeing the blood from its excess of serum, I should re-establish the equilibrium of the circulation, and facilitate the working of the pulmonary apparatus.

Drastic purgatives—purgatives which quickly produce copious serous evacuations, and have for that reason been termed hydragogues—best fulfil this urgent indication. I therefore gave the compound tincture of jalap with forty grammes of hollands. Under the influence of the first brisk purging, the anasarca was sensibly diminished on the following day. At my second visit, I repeated the same prescription, and obtained from it a still more decided effect. The hollands was administered some days later in the same manner: before two weeks had elapsed, the dropsy had completely disappeared, and there was no longer any difficulty in breathing. I was then enabled to verify much more easily than I could at the date

of the patient's admission, the physical and local signs of her cardiac affection.

Under the influence of a class of remedies which the old physicians called *panchymagogues*, that is remedies which purge offending humours from the blood, within from forty-eight to seventy-two hours, I got rid of the extensive anasarca which had occasioned such formidable symptoms. I averted imminent death, which was the sole result which the nature of the case allowed me to aspire to accomplish; for unfortunately, I could do nothing to cure the organic lesion, the basis of the disease.

Having realised this terrible fact, I went on giving the diuretic wine of the Hotel-Dieu, and, after a time, bitters.

Of all the hydragogue remedies which I have ever employed, the most powerful is that known as the "*vin diurétique de l'Hôtel-Dieu*," the formula for preparing which I devised, and which is as follows:—

Take of:—

White wine . . .	750 grammes;
Squill bulbs . . .	5 grammes;
Juniper berries . . .	50 grammes; and
Foxglove leaves . .	10 grammes.

Macerate them together for four days.

Then add of:—

Acetate of potash . .	15 grammes.

Filter.

This wine, which I have employed for many years, and which has been accepted by my colleagues according to my formula which I have now given you, is generally borne well by patients: to it, both in my hospital and private practice, I owe apparent recoveries from affections complicating diseases of the heart—complications for the relief of which there seemed nothing to be done.

Freed from danger for the time being, the patient soon found herself sufficiently well to ask for her dismissal from the Hôtel-Dieu: the only discomfort which remained was some shortness of breath, an inevitable consequence of her disease of the heart. In about six weeks, she came back in a condition exactly similar to that in which she was when first received into the hospital.

This relapse alarmed me all the more that I too well knew that such complications are liable to return, and must ultimately be

beyond the resources of art: a time will come, when—if I may so express myself—the cup being already full to the brim, a single drop will cause it to overflow; in short, I knew that this woman was doomed within a short period to sink under her disease, and that she would probably be carried off by the symptoms which affected the general economy. The success of my treatment had, however, been so conspicuous on the first occasion as to constrain me to resort to it a second time. I again employed the same means; and on the third day, the anasarca had again disappeared, and the breathlessness had again ceased. The patient asked for food, and complained that she had too little to eat; but from the occurrence of a new complication I was prevented from satisfying her appetite.

The diarrhœa produced by the tincture of jalap and diuretic wine continued. That, however, did not give me any anxiety, as I thought that the flux which I had produced with a view to remove the dropsy would also prevent its return. The event was not in accordance with these anticipations, for although the diarrhœa continued, the anasarca steadily increased, till it was as great as when the patient came into hospital. It was no longer possible to recur to the treatment which had been so marvellously successful on two previous occasions, for the administration of drastics and diuretics would have necessarily increased the irritation of the digestive canal, and augmented the disorders of the nutritive function upon which evidently depended the third appearance of the dropsical symptoms. Clearly, the first indication was to modify the pathological state of the intestinal canal: that indication having been fulfilled, there would be ground to hope that the dropsy might be advantageously dealt with by acting on the kidneys or skin, the secretion from which organs might come in the place of that from the intestinal canal.

Subnitrate of bismuth and prepared chalk were first given separately, and then in conjunction, but without any beneficial result. Nitrate of silver given by itself to the extent of 10 centigrammes in the course of the day, in 10 pills, and the nitrate of silver in combination with opium, succeeded no better. The diarrhœa, in place of becoming less, became more profuse. I then employed *hydrargyrum cum creta,* (mercury killed in chalk) a preparation taken from the English Pharmacopœia, where it is called "grey powder:" while, like calomel, this preparation is a purgative, it is also, like it, when

administered in a particular manner, an excellent alterative, a modifier of the state of the intestinal canal, very useful in certain kinds of diarrhœa. The patient took ten centigrammes (about two grains) of grey powder on the first day; and from that day the intestinal flux moderated. Next day, I added three drops of laudanum to my prescription, ordering them to be taken immediately after the grey powder. Under the influence of this medication, the number of stools in the twenty-four hours decreased from seven or eight to two.

This beneficial result was obtained, but the dropsy continued: as it was no longer possible to excite the intestinal, I calculated on the renal secretion. As, however, the substances which act on the kidneys are apt to irritate the intestines, I was afraid that if I administered them internally, I might overturn the therapeutic platform which had cost me so much trouble to erect. I consequently resolved to apply diuretics externally, a practice of which I have had experience during the last twenty years, and which, during that period, has often rendered me signal services.

I cause a strong decoction to be made of squill bulbs and digitalis leaves; or, I take from 100 to 150 grammes of squills and the same quantity of the tincture of digitalis and mix them with two thirds of water. Flannels soaked in this mixture are applied to the abdomen and thighs of the patient, and the flannels are covered up by a large wrapper of oiled silk. By persisting in the use of this measure, a very abundant supply of urine is often obtained. This is what you saw in our patient: in her, too, the diuresis led to resolution of the dropsy. Having a third time got quit of her symptoms, she felt herself sufficiently well to leave the hospital.

You see therefore that in this woman, the diarrhœa which I brought on for the purpose of getting rid of serious symptoms involving danger to life, became in turn a cause of exactly similar symptoms, and that I had consequently to contend against it. In another woman, whom you have likewise seen in our clinical wards, suppression of the usual intestinal flux was the cause of death.

This patient, like the first, came into our wards with an affection of the heart characterised by palpitation of the heart, with irregular frequency and inequality in the arterial pulse. On auscultation, I heard, at the apex of the heart, a sawing murmur accompanying the first sound, continuing during the short interval between the two sounds, and prolonged till the second sound. I diagnosed contrac-

tion with inadequacy of the mitral valve, and hypertrophy of the heart. The precordial dulness on percussion extended beyond its normal limits.

The patient told me that for more than two years she had never been without diarrhœa; but she added, that she never was so well as when she had abundant diarrhœal evacuations. Not paying much attention to this specialty, and not placing implicit confidence in the woman's statements, I endeavoured to moderate the intestinal flux. My attempt was successful; but I bitterly regretted having made it, for great disturbance of the circulation soon supervened, and three days after the cure of her diarrhœa, this poor woman died.

I am convinced that the profuse secretion from the intestinal surface was a discharge which protected the patient from the congestions and dropsies which usually accompany cardiac affections. As soon as I perceived the untoward symptoms occasioned by my inopportune medical intervention, I tried to restore the critical evacuation which I had imprudently checked: my attempts were unavailing. The case of the other patient of whom I have just been speaking would tend to show, that artificial critical evacuations are very far from being satisfactory substitutes for the evacuations which arise spontaneously.

I have gone into these details to show you, how difficult it is to lay down general rules of treatment: to show that that which suits some cases is unsuccessful in others—that therapeutic measures of benefit in given circumstances may fail subsequently even in the same subject—and that we must often seek to attain the same object by different means.

Gentlemen, I have already told you that affections of the heart expose physicians to the risk of making many mistakes—in this sense, that there is nothing so difficult as to form a prognosis of even approximate certainty as to their ulterior progress. I now add, that although it is generally easy from the perfection which in our day has been attained in auscultation and percussion, to ascertain the existence of heart diseases, their diagnosis still sometimes presents great difficulties. This may arise from the structural changes not manifesting themselves during the life of the patients by the physical phenomena which generally characterise them, as is proved by cases reported by Stokes of Dublin. And sometimes, as has been remarked by Dr. Beau, the examination of the dead body confutes our too precise diagnosis of a cardiac affection.

Of this I only wish to lay before you two examples. Let me speak in the first place of a man in whose case I showed the morbid anatomical parts in this theatre. The patient was suffering from hypertrophy of the heart of old standing, and I thought that there also existed insufficiency of the mitral valve with contraction of the auriculo-ventricular opening. He had also had slight hemoptysis. Some of the sputa were tinged with blood: others were black and viscid, while others again were frothy and bright red. Finally, great general anasarca and ascites were added to the already serious complications. The patient died three days after his arrival in our wards.

On opening the body, I found that there was a notable increase in the volume of the heart. This hypertrophy involved the left ventricle, which was also dilated. I detected nothing abnormal in respect of the orifices, except slight thickening of the mitral valve, both segments of which, however, seemed to play perfectly well, there being neither valvular insufficiency, nor any appreciable contraction of the auriculo-ventricular opening. One of the sigmoid valves of the aorta was ossified at its base, but it nevertheless adequately performed its office. There was, therefore, neither valvular insufficiency at, nor contraction of, the aortic orifice. The aorta was somewhat dilated, and its walls presented incrustations similar to those upon the sigmoid valve.

The reasons which influenced me in forming my diagnosis—insufficiency of the mitral valves and contraction of the auriculo-ventricular opening—seemed to me to be the natural inferences from the signs and symptoms now stated.

When, as in our patient, there is only hypertrophy of the heart with dilatation, and no serious lesion of the mitral or tricuspid valves, the circulation generally remains regular.

So it is also, when the sigmoid valves of the aorta are insufficient, a condition which is indicated by the bellows-murmur accompanying the second sound of the heart, heard at the base of the heart, prolonged through the arch of the aorta; by a remarkable vibratory character in the radial pulse; also, by greater force and fulness in the beat of the large arteries, such as the carotid, humeral, and emoral.

The absence in our patient of the physical signs which characterise insufficiency in the aortic valves, although, in spite of the absence of bellows-murmurs, I detected all the phenomena which

indicate lesions of the auriculo-ventricular orifices, viz. very great irregularity in the pulsations, also general anasarca, and a pulse, which besides being intermittent, was so exceedingly feeble as to make it impossible to count the number of the beats. By all these signs, I was led to my diagnosis.

The autopsy proved my diagnosis to have been wrong; and showed that the symptoms observed during life, that the excessive disturbance of the circulation entirely depended on what Dr. Beau has called "*asystolie*"—want of contractile power. I had certainly attributed to this *asystolie* the absence of the bellows-murmur characteristic of lesion of the auriculo-ventricular orifice; but I believed that it did not proceed from impaired muscular contractility of the heart, but from resistance to this contractility by an obstacle situated at the orifices. I based my opinion, I repeat, upon the presence of phenomena which I have described to you, and which are seldom associated with mere increase in the volume of the heart.

There can be no doubt that I committed an error in my diagnosis; but I question whether it was an error which it would have been easy to have avoided. I confess that were a similar case to occur to me, I should be equally embarrassed, and should probably fall into the same error. In reality, the matter is not of much importance in a practical point of view: and here let me repeat a remark which I have already made, that the exact seat of a cardiac lesion is a study more interesting than useful.

Apart from the reasons which I have mentioned, the occurrence of pulmonary apoplexy in our patient was an additional reason for believing that there was a lesion at the auriculo-ventricular opening, because it is in cases in which such lesions exist that pulmonary hemorrhage most commonly supervenes.

The attack of pulmonary apoplexy was slight. At the autopsy, we only found a very small clot, about the size of a pigeon's egg, situated at the posterior part of the left lung. This accounted for its presence not having been revealed during life by auscultation, and explained why we had only heard some mucous subcrepitant râles unaccompanied by bellows-murmur or dulness.

The patient had had albuminuria; and his kidneys presented all the characters of congestion. In this, there was nothing extraordinary; for, as you are aware, there is nothing more common than albuminuria in the last stage of disease of the heart. I say *albuminuria* and not *Bright's disease*, which is a very different affection.

Albuminuria is an expressive symptom met with in a great many diseases, such for example as typhoid fever, small-pox, scarlatina, and diphtheria—a symptom which may be transitory, and relate either to a temporary state of the kidneys or of the blood. Bright's disease, again, in which albuminuria is the predominant, or if I may so speak, the specific symptom, is characterised by a structural change, irremediable and more or less profound, in the kidney. The presence of albumen in the urine of individuals affected with diseases of the heart probably depends upon passive hyperemic congestion, on engorgement, on hypostasis, which may take place quite as readily in the kidneys as in any other viscus, their vascular tension allowing transudation of the most liquid part of the blood, or, in other words, of the serum. There takes place in the urinary apparatus something analogous to that which takes place in other organs, in the cellular tissue, in the serous cavities in particular, where under the influence of the impediment to the venous circulation, we see passive congestions occurring, which give rise to dropsies.

The second example which I wish to bring before you is the case of a woman who was received into the hospital with all the local and general symptoms of an affection of the heart. There existed an abnormal amount of dulness in the precordial region; and a bellows-murmur, having its maximum intensity at the apex of the heart, was heard, instead of the click of the valves. The pulsations of the heart were unequal in force, and the pulse was so quick that it could not be counted. There existed greatly embarrassed breathing and œdema of the extremities. The general symptoms yielded to the treatment which I adopted; but the serious character of the local phenomena continued, excepting that there was a diminution in the rapidity of the pulse. The diagnosis which I had ordered to be inserted in the descriptive paper on the bed [*la feuille d'observation*] was—"valvular insufficiency, contraction of the auriculo-ventricular opening, and hypertrophy of the heart." This was also the diagnosis of those who were in the habit of following me in my hospital visit, and who had on many occasions, at intervals of some days, attentively examined the patient, as I had also done. I had no idea that I was mistaken; when, upon one occasion, (after having for some time discontinued to auscultate the heart), I was surprised not to find the bellows-murmur, previously so manifest. For eight days, there was no return of this murmur, and the sound of the valvular

click was only a little duller than in the normal state. There was, moreover, no return of the general symptoms.

I certainly could not pretend to have cured the cardiac affection: I was well aware that when lesions of this kind attain a certain point, they do not improve: their remaining stationary is the most favourable result which can occur. To a certain extent, therefore, I was mistaken: the bellows-murmur accompanying both sounds of the heart very evidently indicated a morbid condition at the auriculo-ventricular orifice, an obstacle to the free play of the valves, which only imperfectly closed the opening, while, at the same time, there was a diminution in the calibre of the opening. There existed a permanent cardiac lesion, which was apparently a certain amount of thickening of the valves. There had been a temporary lesion, probably constituted by the vegetations, the presence of which upon the surface of the valves has been pointed out by Laennec, Professor Bouillaud, and others.

Gentlemen, there can be no doubt that it is impossible absolutely to diagnose such lesions, but we may have good grounds for suspecting their existence. It is my opinion, therefore, that in our patient fibrinous concretions existed, which under the influence of a condition, probably inflammatory, were deposited upon the already diseased valves, concretions such as are deposited—to use Dr. Bouillaud's happy comparison—upon the rods with which blood is whipped to separate its fibrin. These concretions interfered with the play of the membranous valves to which they adhered, and also narrowed the calibre of the auriculo-ventricular orifice, causing the bellows-murmur which we heard. The disappearance and non-reproduction of the bellows-murmur must be ascribed to the opening being rendered free by removal of the partial obstruction, its gradual solution, and no new fibrinous deposit having taken place. I am unable otherwise to explain the production and cessation of the physical phenomena observed; but I admit that very serious objections may be urged against the validity of this explanation.

This is no doubt an exceptional case; but it is not unique in the annals of science. Such of you as have attended my clinic for some time must have seen patients presenting all the local and general symptoms of a very advanced cardiac disease—great anxiety, extreme depression, purple face, rapid and irregular pulse—recover from these symptoms, which admitted of no other explanation than a temporary obstacle to the circulation caused by polypiform concretions.

Irrespective of symptoms resulting from the disturbance of the circulation through the cardiac cavities, there are others of a still more serious character which are generally consequences of fibrinous concretions in the heart or vessels:—I refer to the symptoms caused by embolia.

The term *embolium*, as you are aware, signifies a foreign body, which, formed in the heart, the arterial system, or the venous system, is launched into the torrent of the circulation, and obliterates more or less completely the vessel in which it becomes impacted. The migration of clots in the vascular system is a fact to which attention was first called by Legroux, or at all events, he was the first to describe their migration in the arterial system. More recently, and particularly since the researches of Professor Virchow, the subject has been studied anew, and in a more complete manner.[1]

Embolia, according to the importance of the vessels which they obstruct, may occasion more or less serious symptoms. Suppose for example, that a fragment detached from the polypiform concretions of which I have just been speaking, be impelled through the aorta into the main artery of a limb, that it be there arrested in its progress, forming a plug by which an obstacle is presented to the passage of the blood, gangrene of the limb will be the consequence. The gangrene will be analogous to that very inappropriately called *senile* gangrene, which is also the result of arterial obliteration. Suppose again, that the embolium stops the circulation in one of the principal arteries of the brain, you can readily understand that the consequences will be much more serious, and may even prove rapidly fatal.

Some years ago, a young married lady whom I was at the time seeing in consultation with my friend Dr. Voillemier felt uncomfortable sensations in the region of the heart, and was afterwards suddenly seized with painful tingling in the fingers. The fingers had a bluish colour, and very soon presented all the appearances of a dry gangrenous affection: fortunately, the gangrene was limited to one of the last phalanges which the patient lost. Eighteen months later, this lady was suddenly struck with complete paralysis of one side of the body, and subsequently sunk with all the symptoms of softening of the brain. The age of the patient made it improbable

[1] Virchow:—Uber die Verstopfung der Lungenarterie. [Froriep's Neue Notizen, 1846.]

that she had had an attack of simple apoplexy, the suddenness of the paralytic seizure, and the nature of the symptoms which had eighteen months previously shown themselves in the hand, led me to the conclusion that on both occasions something similar had occurred. On the first occasion, an embolium having obliterated an artery of the hand, caused partial paralysis of the finger; and on the second occasion, an embolium penetrated the arteries of the brain and produced softening—a sort of cerebral gangrene, of which hemiplegia was the characteristic manifestation.

Nearly at the same time, a friend, quite a young man, was carried off by an attack of paralysis, which supervened under circumstances similar to those now described: the hemiplegia with which he was suddenly seized came on abruptly in the course of an attack of rheumatism of the heart.

Two years previously, one of my colleagues was sent for to Bourges, to see a patient who had rheumatic endopericarditis: and in whom had suddenly supervened gangrene of the great toe accompanied by coldness and purpling of the integuments. After death, which was not long in occurring, the popliteal artery was found completely obliterated by a clot.

Similar facts have been recorded in different medical repertories. Dr. Worms has reported a case of acute endocarditis, followed by gangrene of the left leg caused by an embolium which had obliterated the trunk of the tibio-fibular artery. The patient was a soldier, 29 years of age. The gangrene was complete, and involved the loss of the limb in which it occurred. The two lower thirds of the leg separated almost without any interference; and six months after that occurrence M. Follin was obliged to give shape to the stump by an operation.

Judging from what takes place in limbs attacked by gangrene, we may conclude that the apoplectic attacks of which I have been speaking may originate in a similar mechanism. As the anastomosing arteries of the brain are so numerous and so large that the common carotid, and even both carotids, may be tied without occasioning either death or cerebral lesion, it is possible that in cases of embolism the apoplectic phenomena are the result, not merely of vascular obliteration, but of a more direct action of the clot upon the cerebral substance.

Be that as it may, the disturbance of the cerebral functions is subordinate to its cause. Let me explain: if an embolium is only

large enough to obstruct partially the vessel in which it is impacted, if it be of so slight a consistence as to admit of being disintegrated and dissolved, the obstacle which it presented to the current of the blood disappearing, the brain will resume its functions; but if, on the contrary, the embolium is large enough completely to plug the vessel, of a sufficiently resisting consistence not to break up and dissolve, there will ensue a real gangrene of the brain, and the cerebral softening may lead to death.

You must remember a woman of 47 years of age whom we had in bed 4 of St. Bernard's ward. She came into the hospital to be treated for disease of the heart; and my diagnosis was—contraction of the auriculo-ventricular orifice with inadequacy of the valves. I also found that there existed ascites and anasarca. The state of liver, which extended beyond the false ribs and seemed to be increased in volume, led me to believe that there was incipient cirrhosis.

After some time, the condition of this woman had improved: the anasarca had disappeared, and the ascites had decreased; when, in the beginning of December, suddenly she felt very acute pain in the right side of the head, and was struck with hemiplegia of the left side of the body. She retained consciousness up to death, which occurred ere long. My diagnosis was:—softening of the brain, caused probably by obliteration of an artery.

The autopsy was made on the following day at my request, and with the greatest possible care by Dr. Ludovic Hirschfeld, a very distinguished anatomist, now professor of anatomy in the medical university of Varsovia. The middle cerebral artery, which presented no trace of ossification, was completely obliterated by a clot of blood, black, homogeneous, and three centimeters in length: the branches going to the right corpus striatum, which was in process of softening, were also obliterated in the same way. All the other arteries were free from obstruction.

I found also serious lesions at both auriculo-ventricular orifices, the valves of which were indurated, adherent, and insufficient. There also existed dilatation with hypertrophy of the heart.

The cirrhosis of the liver which I had diagnosed certainly existed; but the disease was in a much more advanced state than I had supposed. The increased volume of the liver was only apparent, the organ being pushed down by the right lung, which was in a very emphysematous state.

Bear also in mind the case of the recently delivered woman who occupied bed 20 in the same ward. The attendants attached to the ward informed us that on the evening before her admission to the hospital she had been struck by paralysis without immediate loss of consciousness: she had been able to say:—"take me to the hospital"—but on the day on which I saw her, she could not reply to any of my questions. I found that there was complete hemiplegia of the motor power of the right side, but no diminution of natural sentient power.

The pulse was frequent and irregular: auscultation of the heart enabled one to hear a bellows-murmur, having its maximum intensity at the apex, beyond the nipple. This murmur was harsh, and the mitral orifice was its probable seat. Connecting those two facts—the lesion of the left side of the heart and the cerebral apoplexy—recalling to mind the beautiful researches of Virchow upon the migration of clots, and reasoning from the facility with which sanguineous coagula form in recently delivered women, and upon the rapidity with which the symptoms were developed in the case now under consideration, I had no hesitation in concluding that I had to do with an embolium situated in the middle cerebral artery of the left side, which had induced softening of the corresponding parts of the nervous centres. The patient, without having had any inflammatory reaction, without regaining consciousness, and without showing any change in the state of the paralysed side, died eight days after her admission to the hospital.

The autopsy fully confirmed my diagnosis. A portion of the brain in front of the left corpus striatum was softened: on the same side, the calibre of the middle cerebral artery, in the situation in which it expands into a dense vascular net-work, was obliterated, only, however, to the extent of two millimeters, by a small fibrinous clot which was yellow, resistant, and did not adhere to the parietes of the vessel: all around this little fibrinous clot, there was coagulated blood, which on the one side became lost in the anastomosing plexus of the middle cerebral artery, and which on the other side terminated abruptly at the origin of the artery of Sylvius. The little central clot which plugged the artery was like a millet seed. There was no lesion of the walls of the middle cerebral artery.

At the bifurcation of the left common carotid artery, there was a small fibrinous clot which also was of the size of a millet seed, which sent three filiform prolongations, cruoric and fibrinous, one into the

common carotid, another into the internal carotid, and the third into the middle thyroid.

The mitral valve was the seat of conspicuous lesions. On the auricular surface of the valve, some adherent to the pericardium, and others so free as to be almost unattached, were warty looking clots of different sizes. Gentlemen, it would be very difficult to avoid seeing that there was a relation of cause and effect between the mitral lesion and the fibrinous clots found in certain cerebral arteries and the accompanying softening of the brain. Observe that the other arteries of the brain, examined with care, presented no intra-vascular coagulation, and that, except in the situation of the softening, the entire brain was normal in colour and consistence. Finally, the similarity of form and the identity of structure of the fibrinous concretions of the mitral valve with those of the artery of Sylvius constituted an additional reason in favour of embolism.

With reference to the softening of the brain, I wish to remark to you, that most frequently the lesion of consistence and nutrition is seated in that portion of the left hemisphere supplied by the middle cerebral artery, or artery of Sylvius. I am not going to attempt to explain this fact to you, for were I to do so, I should only be entering upon an anatomical discussion of doubtful value.

I would merely, in relation to our patient, remind you that my colleague, Dr. Broca, in his interesting memoir on aphemia, or the loss of articulate speech, has localised the function of speech in the posterior part of the third frontal convolution of the left side.[1] Let me also remind you that cerebral softening is most frequent on the left side, and in that portion of the brain which is supplied with blood by the artery of Sylvius. We are led by a consideration of these facts to see that there may be a relationship between an embolium, the seat of cerebral softening on the left side, and aphemia, or *aphasia*, as I have proposed to call the affection. This is a point to which I propose to return in future lectures.[2]

To revert to the subject more immediately before us:—it is now a well established fact that migratory clots of a certain volume may, when lodged in the brain, or in other parts of the body, lead to softening or to gangrene: these results do not always take place

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[1] Broca:—*Bulletins de la Société Anatomique.* 1861.

[2] See the lecture on Aphasia p. 218 of the First Volume of the New Sydenham Society's translation.

when the clots are so small as to be only stopped in their passage through the capillaries. The consequent phenomena may then be of another kind. Capillary embolia seldom cause partial, very circumscribed gangrene, but often give rise to numerous ecchymotic spots, small parenchymatous abscesses, and secondary deposits of fibrin, to which the name of visceral stuffing or *infarctus* has been given. The capillary embolia are the result of the breaking up of the clots, and disintegration of the fibrin deposited on the cardiac valves; or, of the spontaneous opening of atheromatous, fibrinous, or purulent cysts of the arteries.

There are some cases in which the starting point of embolia is *ulcerous endocarditis*, which at other times gives rise to symptoms of general poisoning, leading to a typhoid state. This is a point which I propose afterwards to develope in lectures specially devoted to the subject.[1] Here, I will only remark that Professor Bouillaud, in his "*Traité des Maladies du Cœur*," has reported a case of gangrenous endocarditis with ulceration and perforation of the aortic valves; but both Professor Bouillaud, and Dr. Gigon (of Angoulême) who quotes the case, have only called attention to the morbid phenomena of endocarditis and to the contraction of the orifice with inadequacy of the aortic valves. Subsequently, Rokitansky, Virchow, Bamberger and Freidreich in Germany, as well as Charcot and Vulpian in France,[2] showed that ulcerous endocarditis may be the starting point of capillary embolism; and that it may also, by introducing morbid elements into the current of the circulation, give rise to symptoms of a general putrid poisoning of the system so closely simulating, as to be liable to be mistaken for, the symptoms peculiar to typhoid fever and severe jaundice.

Ulcerous endocarditis is more frequent in the left than in the right side of the heart: such at least is the conclusion arrived at from the cases collected by Bouillaud, Virchow, Bamberger, and Freidreich: Charcot and Vulpian, however, have published a case in which the valves of the right side of the heart were affected. To explain the symptoms of poisoning which

[1] See the lecture on Ulcerous Endocarditis.

[2] Charcot and Vulpian:—Cases of numerous Fibrinous Tumours containing Puriform Matter. [*Comptes rendus des Séances et Mémoires de la Société de Biologie: Paris*, 1854, *p.* 189.]—Memoir on Fibrous Cysts containing Puriform Matter in two Cases of Partial Aneurism of the Heart. [Second Series: Vol. I, 1854, p. 301.]

occurred in that case, it is necessary to admit that the products of the ulcerative action in part had passed through the capillary vessels of the lung: such is the hypothesis enunciated by Drs. Charcot and Vulpian. But though we grant that the blood may be poisoned by the products of ulceration, it is not necessary to assume that they travel over the whole vascular system: it is sufficient that the blood in the right side of the heart come in contact with these products, to be changed to such an extent as to cause general poisoning.

Just as the coincidence of an organic lesion of the heart with apoplexy leads us to infer that there is an embolium in the brain, so does the coexistence of cardiac symptoms with a typhoid condition not depending on dothinenteria, justify the supposition that there is ulcerous endocarditis.

I propose to study with you the subject of *venous embolism*—very common in cachexic conditions and in the puerperal state—when I come to lecture on *phlegmasia alba dolens*.

LECTURE LII.[1]

ALCOHOLISM.

Symptoms of Alcoholism Referable to the Nervous System.—Delirium Tremens.—Influence of the Habitual Use of Alcoholic Stimulants on the Progress and Treatment of Diseases.—Successive and graduated [hierarchisés] Symptoms caused by Alcohol in its Passage through the Organism.—Lesions of the Stomach and Subsequent Lesions of Organs in the Cycle of the Venous System.—Lesions of Organs in the Cycle of the Arterial System, the Nervous Centres, the Kidneys, &c.—Steatosis and Cirrhosis.

Gentlemen :—To-day, I propose to speak to you on the subject of Alcoholism.

Without concealing any of its difficulties—because I fully recognise their existence—it may still be useful to state to you my views upon this problem, one of the most difficult in pathology. If it be necessary to impress upon young physicians the classical precepts of their profession, it is not less useful, at rare intervals, to make with them more perilous scientific excursions.

The affections of the nervous system, of which I have given you a summary and necessarily incomplete history, present in a less degree than the affections of other organs, but still they do present, a certain degree of uniformity and definite character in their symptoms. To say that a man is *epileptic* is to characterise the individual, the symptoms from which he suffers, and the future which is in store for him. To say that a woman is *hysterical* is to place her in a more vaguely defined class. But subordinate to these grand divisions of

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[1] Lectures XXXIX—LI will be found in the First Volume of the Translation of the New Sydenham Society, in the order stated in the "Notice to the Reader" prefixed to the Second Volume. Translator.

nervous affections, how many minute divisions are there which escape even a vague classification!

When, in place of obtaining the elements of our definitions from an interrogation of symptoms, we go back to the causes which have provoked, awakened, or maintained derangements of the nervous system, we attain to a more intimate knowledge of the nature of the disease: but here, as in everything else, the more we leave the surface, the more do obscurities increase, and outlines become uncertain.

You all know, Gentlemen, and unprofessional persons know as well as you do, that alcoholic preparations produce obvious effects upon the organism, and that, in the first instance, they show a predilection for the *cerebro-spinal system*, although at a later stage, the other great systems are variously affected, as I shall endeavour to explain to you. You are also well aware that unfortunately there is a great prevalence of dissipation among the class from which the hospitals chiefly derive their patients. This fact constitutes an etiological element which you will too often encounter complicating diseases, disturbing their evolution, and impeding convalescence.

And yet, till very recently, the custom has been to consider the study of the peculiar effects of alcoholism as belonging only to physicians specially devoted to mental diseases. I accept, but never without profound regret, the artificial limits imposed by the administrative exigencies of practice, or by personal deficiency; but in the matter which I have now brought under your notice, I cannot resign myself to any similar conventional arrangement. Extreme cases, it is true, may only be accessible to observation in the wards of a lunatic asylum, but it is otherwise with cases of moderate severity—with effects more limited in character and not culminating in insanity. Alcoholism belongs to the same category as many other cerebral affections. There is an insensible gradation of cases between the attack of apoplexy consequent upon which the mental powers have fallen to the lowest form of dementia, and the apoplectic seizure after which there only remains an almost imperceptible amount of hemiplegia: but in this gradation of cases, you will be unable to appreciate the value of the symptoms which chance has brought under your notice, unless you keep in view the two extremes of the progressive series.

This statement is still more applicable in respect of alcoholism. In the first instance, the effects of alcohol are within the domain of

physiology: taken in small quantities, alcohol has its right and proper place in the aliment of the body: but taken in excess, it either leads to formidable maniacal excitement, or to the pitiable spectacle of acquired idiocy. You will admit, Gentlemen, that within this vast domain, it will only be possible for me here to point out some landmarks.

Alcoholic compounds used in what I may call physiological quantities are unquestionably beneficial to the human subject in a state of health: there are also cases in which they may be useful in a state of disease. I do not require to recapitulate the circumstances under which their administration is appropriate in health or disease. And yet what useful knowledge there is to be learned in relation to that subject! You have often seen me prescribe wine in tolerably large doses in adynamic fevers, and during convalescence from inflammatory affections which have left great depression behind them.

Fermented liquors constitute more than a complement to alimentation: they likewise fulfil another class of indications. For a long period, however, their employment in medicine was proscribed as a perilous enormity; but latterly, by one of those reactions of which therapeutics offers so many examples, there has been no shrinking from giving them with extreme hardihood. Without running any risk by following these aberrations, we may profit by daring trials from which physicians of most reliable character have not shrunk.

The clinical experience of the present day teaches us that even patients suffering from inflammatory affections tolerate fermented drinks in quantities which formerly one would have been far from suspecting. Though it has not been demonstrated that they do as much good as was hoped by the physicians who recommend them, it is certain that they do not produce the injurious consequences dreaded on theoretical grounds. It is an additional proof in support of that law, upon which it is impossible to insist too often, that during the existence of a pathological state, the action of toxic medicines is profoundly modified. Just as you have observed opium pushed with impunity to doses which may be called imprudent in refractory diseases, so have you seen large doses of alcoholic liquors borne without any bad consequences by patients suffering from various affections of the nervous system.

The data which I am about to give are not laid before you as examples to follow; but as facts calculated to assist in the study of the toxic effects of alcohol.

Pathologically, as well as physiologically, it is important to distinguish between the healthy and the sick individual, so that we may not attribute to the one as an incontestable verity, an effect which is a reality only in respect of the other.

When a man in the plenitude of health gives way to alcoholic excesses, more or less frequently, and to a more or less serious extent, he experiences effects which differ in form and degree. If the same individual during self-induced intoxication falls into sickness, he forthwith, in consequence of the incident disease, stands in a different relation to the toxic agent: neither the susceptibility, the phenomena, nor the consequences are necessarily the same as before the pathological change took place. By bearing in mind this fundamental distinction, you will be saved from more than one source of confusion, and get the key wherewith to explain many mistakes.

In the hospital, it is only on rare occasions that you have an opportunity of observing the effects of excessive alcoholic potations upon persons who are not affected by disease. Here, drunkenness appears as one of the remote and often doubtful antecedents of the affections which it engenders; and is a matter of sharpened memory and not of direct observation. At other times, it explains the tendency which patients have to become delirious in the course of diseases in which delirium is not an essential symptom: and it induces a predisposition to disturbance of the nervous system; but its intervention in this class of cases is more conjectural than in the other. You have seen, however, some cases, which I have been careful to point out to you, of alcoholic delirium uncomplicated with any other affection.

Drunkenness in its first degree presents you with a miniature picture of the symptoms of the more advanced stages of ebriose poisoning: on the one hand, there is gastro-intestinal disturbance; and on the other, there are disorders which are either nervous and secondary, or purely sympathetic. The mouth is pasty, the tongue is dirty, the stomach is surcharged, and there is nausea: the head is heavy, the senses are either in an excited or obtuse condition, and there is giddiness verging upon syncope. Cerebral excitement gives place to invincible depression, and the sleep reminds one of apoplectic stertor.

The fit [*l'accès*]—let me use that word—is of short duration, but it leaves behind it a state of discomfort which is more prolonged,

and which, in accordance with the temperament of the individual, dominates one or other order of symptoms.

Up to that point, the individual, to a certain extent, retains his individuality. He is good tempered or irritable in his cups [*il a le vin bon ou mauvais*] to use the vulgar phrase, according to the tendencies of his character: he is more or less out of sorts, or unwell, according to the nature of his constitution.

In the second degree of drunkenness, the inebriated man is a sick man [*l'évrieux est un malade*]. The perversion of disposition is so great that the man is no longer himself: the symptoms become developed in the usual way, and are no longer obedient to diversity of temperament. The delirium then assumes a well marked character, the nervous disorder takes a definite form, and the totality of the symptoms is summed up in the name—*delirium tremens*.

The delirium has a sufficiently special character to enable the experienced practitioner to recognise it, without requiring him to know the previous history of the case. The physicians who have made mental alienation a special study have described *delirium tremens* in a very masterly manner, and to their works I refer you for a complete description of the disease. It will be sufficient for the present, that I point out to you some of its most decisive signs.

In *delirium tremens*, the delirium is characterised by restlessness and mental perplexity, even in cases in which there is extreme violence. The excitement results from fear; for terror itself has its times of audacity. The patient, pursued by hallucinations (particularly of the sense of sight), threatened by assassins, attacked by robbers, is a prey to a thousand miseries. He wishes to start on a journey to some place—it matters not to what place:—his desire is to get away from himself: he folds his clothes, and escapes by every possible way not interdicted to his vagabond impulses. In the midst of his disorderly excitement, he still retains the power of collecting his thoughts by the powerful effort of a dominant will; but this recovery of reason is brief, and ere long the patient returns to his vagaries.

Are you not struck, Gentlemen, with the great similarity between *delirium tremens* and the delirium which supervenes in the course of many serious diseases? On the one hand, there are hallucinations which are almost exclusively confined to the sense of sight: then there exists the possibility of momentarily suspending the delirious

conceptions: and lastly, there is the strange propensity to pack up for a journey. I refrain from pursuing this parallel any further, though it is instructive in more than one respect. I ought, and it is my wish, to confine myself to the briefest indications.

While the intellectual faculties are in this perverted state, that portion of the nervous system which does not preside over the mind is equally stricken. The most constant and the most conspicuous of all the nervous perturbations in this disease is trembling.

From a semiological point of view, trembling is one of the phenomena most accessible to investigation, and yet the most difficult to describe categorically. This is an obscure subject: trembling is a symptom of uncertain meaning, and its import is all the more doubtful, that a tendency has been shown to assign to it a value which it does not possess.

It is a very prejudicial error to rely on a mere pathological phenomenon, as if it were a diagnostic sign. I am quite aware that there is senile trembling, mercurial trembling, and many other kinds of trembling. But to classify after this fashion the different kinds of trembling, as if they were so many distinct unities, is to approximate to ontology more than to reality. In point of fact, trembling is one of the signs of old age, and of mercurial poisoning, as well as of alcoholism. To the latter, it does not exclusively belong, as a pathognomonic phenomenon.

Subject to this express reservation, alcoholic tremor has some peculiarities. In its most marked form, it is a sort of general fremitus. Lay your hand upon the shoulder, and you will feel the patient vibrate so to speak; but this description is not always applicable. It is sometimes characterised by muscular *subsultus*, the trembling being sometimes so convulsive as to make the step hesitating, and the prehension of objects almost impossible: you may perceive it either to cease or to continue under the influence of excessive maniacal excitement, which latter occurrence is the reverse of what takes place in the trembling of paralysis. You will observe that sleep does not suspend as certainly as it interrupts the choreic movements. Be that as it may, Gentlemen, bear in mind that trembling is not necessarily the companion of alcoholic delirium, when that delirium comes on in persons otherwise in good health; and that it is far from being constantly met with when drunken delirium [*délire ébrieux*] makes its first appearance under the influence of a disease.

I do not now speak to you of the gastric symptoms relegated to the second phase of the disorder, or possibly entirely annulled by the magnitude of the nervous symptoms peculiar to *delirium tremens:* but we shall forthwith meet with them again and in chronic alcoholism, in which they are aggravated by repetition.

The crisis of *delirium tremens* is violent, but, relatively, it is short; and it is seldom fatal. After many therapeutic attempts—too often fortunate not to awaken some doubt—many physicians have come to restrict themselves to the expectant system, save in exceptional cases.

When the fit [*l'accès*] is terminated, all is not ended. It is an old and a true proverb, that "he who has drank will drink" [*qui a bu boira*]. The drunkard who has come through an attack of *delirium tremens* is as seldom cured of his passion for alcohol as is the gambler for his passion for play, and is in general excited to new debauches. Dipsomania is more frequently the sequel than the antecedent of a first attack of the delirium resulting from intoxication. The occasions on which the delirium shows itself recur; and the intoxication, which at first was acute, ultimately becomes chronic.

This, however, is not always the manner in which chronic drunkenness begins. While it is sometimes, as I have stated, the result of a repetition of acute fits, at other times, it originates in successive gradual poisoning, producing no shock, and arising from the habitual ingestion of alcohol in slowly increasing doses. Chronic alcoholism has been recently the subject of scientific researches; and you are no doubt acquainted with the remarkable description of it which has been given by Magnus Huss.[1] You know in how excellent a spirit of observation and method he has classified phenomena, the etiology of which he has so well elucidated. He has placed in one category *nervous disorders limited to different encephalic affections*, and in the other category, *disorders of the digestive system* resembling other cachexiæ. You are aware that within this vicious circle, so often met with in medical cases, perturbation of the nervous system contributes to impair nutrition.

In his truthful, lively, graphic picture, the scientific professor of Stockholm exhibits chronic alcoholism—which derives its charac-

[1] Huss (Magnus):—Chronische Alkoholskrankheit. *Aus dem Schwedischen übersetzt von Gerh. van dem Busch.* Stockholm, 1852.

teristics from its very chronicity—assuming numerous forms all of which, if you will pardon the use of an energetic popular locution, are really the small change [*la monnaie*] of the acute stage of *delirium tremens*. The mental phenomena are the same—they develope themselves more slowly, but their change of type is only apparent. Slacken the tumultuous pace of the fancies which jostle and caper in the maniac's brain, and, although you do not thereby effect any essential modification, you will completely alter the aspect of his delirium. For the disgust, the repugnance at food which characterises febrile anorexia, substitute passive indifference, absence of appetite, the gastric state of chronic alcoholism: in place of disturbance of vision, or the changing hallucinations of delirium tremens, there are confused perceptions, *muscæ volitantes*, cloudiness, fogginess, and transient flashes of false light.

But Dr. Huss made his observations under conditions of so special a character, that you require to exercise some reserve in drawing general conclusions from them. He studied alcoholism in a population addicted to the gradual abuse of alcohol, ill-fed, subsisting almost exclusively on vegetable diet; and which, under peculiar conditions, suffered the consequences of inveterate tippling habits. This population had no compensatory intervals of relative sobriety between the fits of debauchery, nor was its daily corporeal waste restored by generous fare. The type of drunkard sketched in so masterly a manner by Huss is that of the drunkard of northern countries: it is not the type of the drunkard met with in France, except among the very dregs of the people. Here, as a rule, intoxication is not gradual and successive, but is generally intermittent—acute during the drunken fit, and suspended during some succeeding days. The description loses its uniformity of application; the symptoms are more adventurous, for even poisoning has its diversities and its adventures.

But there is a question of still greater delicacy to which I wish to direct your earnest attention. Not only is chronic alcoholism far from showing itself with the aggregate of its characteristic signs, but as a consequence even of the intervals which I have been pointing out to you, it may exist, and yet never reveal itself by any phenomenon: it is this latent alcoholic saturation which is specially interesting to the physician, because it breaks forth at decisive moments. If an acute disease attack the individual who is thus saturated, the alcoholism explodes, and throws into the scale the

formidable odds of its unforeseen delirium, or of an ataxia disproportionate to the actual disease.

When this is the programme—and in our country it is a very common programme—have we not at least indications to guide us in forming a retrospective diagnosis, a matter of so much difficulty even to physicians who understand its importance?

Since the time of Dupuytren, it has been asked:—To what extent is the high delirium of patients upon whom amputations have been performed, a manifestation of latent alcoholism? This question has been answered in different ways. Physicians have had no doubts in respect of their department. A man, during the course of a moderately severe attack of pneumonia, in which the fever does not run high, is struck unexpectedly, and almost quite suddenly, with delirium: by what sign are we to recognise the true cause of this perturbation? I wish that I could furnish you with decisive signs; but I have not them to give, and science does not possess them. In your diagnosis, rely neither on the trembling, nor on the nature of the delirious fancies. Both are met with in patients whose antecedents are beyond suspicion. You will either be forced to proceed to a sort of inquisition into the habits of your patient, an inquisition always beset with sources of uncertainty and error; or you will have pretty nearly to trust to uncertainties and erroneous data, which it has been wished—but it has not been known how—to banish from medicine.

When you have summed up the information which you have collected, when you have deliberately weighed the import of the symptoms, and formed your opinion—what are you to do? The classical rule directs the physician to take into account the habits which the patient has acquired, and give him, within judicious limits, the excitant for which he has the artificial craving. If this treatment is successful, the case serves as a confirmation of the rule.

Unfortunately, experience is more complex than it seems to be. At the beginning of this lecture, I dwelt upon the action of alcoholic stimulants in the course of acute diseases; and you have seen the length to which that system of incendiary therapeutics may be carried with impunity. I guarded you against adopting conclusions which are often imperfectly justified. In such cases, take your indications less from the habits of the patient when in health, than from the phenomena of his disease: do not have recourse to alcoholic

stimulants, unless they seem to be required by the symptoms actually present.

Gentlemen, in the preceding picture I have only presented the effects of alcohol upon the nervous system and its injurious influence upon innervation; but that is only an incomplete history of its symptoms. Thanks to modern researches, we can now trace what may be called the physiological history of alcoholism, and follow the alcohol in its passage through the organism. I am also going to show you how alcohol acts upon each of the organs—how it produces lesions of them the sooner it reaches them, the longer it is in contact with them, and the more fragile and impressionable they are.

It is because the encephalon is fragile and impressionable that it is the first organ to feel the effects of alcohol, as well as the organ which feels them in the highest degree; and it is because these effects at once manifest themselves by an assemblage of striking symptoms that the history of alcoholism has for so long a period been cut short. There has been a greater disposition to describe the symptoms immediately resulting from the absorption of alcohol, the early manifestation of which left no doubt as to the relation between the symptoms and the alcohol; while the more remote effects produced by the contact of alcohol with the tissues has either been left in the shade, or quite misunderstood.

The symptoms which I am now about to describe to you in a succinct manner are precisely due to this contact, and they take their graduated ranks [*ils se hiérarchisent*] so to speak, according to physiological laws. Scarcely are alcoholic substances ingested than they exercise an action upon the stomach, and then, in less degree upon the intestines: absorbed to a great extent by the veins of the stomach, they pass through the portal system into the liver, into the right side of the heart, and thence into the pulmonary artery. These substances pass from the lesser into the greater circulation, and there act successively upon the walls of the arteries, and upon the tissues of different organs. It has been shown by the recent experiments of Maurice Perrin, Ludger Lallemand, and Duroy,[1] that alcohol is not decomposed in traversing the organism, and that it does not resolve itself into secondary products such as

[1] Ludger Lallemand, Maurice Perrin and Duroy:—Du Rôle de l'Alcool et des Anesthétiques dans l'Organisme.

carbonic acid and aldéhyde, as used to be believed; but that during the whole period of its sojourn in the tissues, it is alcohol, and as alcohol acts on them. Such being the case, it is evident that it will act the more energetically, the greater the quantity of it in a given organ; in the first place, according to whether that organ receives it before or after the alcohol has passed through the lungs —for a great part of the alcohol taken into the system is thrown off by pulmonary exhalation; and in the second place, according to the degree in which the organ is vascular.

The liver is the first organ in the track of absorption, and the whole quantity of alcohol absorbed traverses it. Moreover, it is not only a very vascular organ, but one in which the blood sojourns a long time for the requirements of the biliary and glycosic secretions. You can, therefore, at once perceive that the liver ought, more than any other organ, to be affected by the alcohol ingested; and such is, as I shall show you, really the case both in acute and chronic alcoholism. But the alcohol alters the condition of the walls of the pulmonary artery, and, consecutively, the tissue of the lungs, just as it alters the state of the parietes of the vena porta.

A very considerable portion, however, of the absorbed alcohol disappears during respiration by exhalation from the surface of the pulmonary vesicles and bronchial tubes. It is, therefore, only a smaller portion, the remaining portion, which affects the other organs, including the nervous centres which feel its influence all the more energetically that the nervous elements, tubes, and cellules, are exceedingly impressionable, as I have already given you to understand. Next come the kidneys, organs which are very vascular, and which, like the liver, preside over an important secretion—a depurative and eliminative secretion. Hence, the kidneys are in prolonged contact with the alcohol: and consequently, we see them frequently and seriously compromised in chronic alcoholism, although less frequently, and sometimes less seriously, than the liver, for the reasons which I have stated; viz. that the liver is traversed by the whole of the alcohol which has been absorbed, while the kidneys only receive that portion which has escaped from being thrown off by the lungs.

As a sequel to this general and purely physiological view of the effects of alcohol upon the organism, allow me, Gentlemen, to enter into some details.

I have led you to anticipate the statement, that the stomach is morbidly affected in inveterate drinkers. There exists a real *gastritis*. It is this condition which explains the anorexia which goes on increasing till at last it becomes absolute—it is this which explains the dyspepsia of drunkards: it is this also which causes the mucous vomiting in the morning to which drunkards are subject—*vomitus matutinus potatorum*—a true gastrorrhœa related to chronic gastritis. This affection is characterised anatomically by a reddish colour and some ecchymotic spots on the mucous membrane, particularly in the neighbourhood of the cardiac orifice and smaller curvature: these are the characters of the first stage of the affection: afterwards, the mucous membrane is thick, and puckered, of a greyish or slate colour, and stained by the pigmentary matter of hematinous deposit: still later, it is hard and friable, and after a time, it undergoes true softening. The glands of the stomach are hypertrophied. In great beer drinkers who imbibe enormous quantities of that fluid, the cavity of the organ may be dilated, and in such cases there is generally thinness of the mucous membrane: usually, the cavity is diminished in capacity from puckering of all its tunics.

If the inflammation, acute or subacute, reach the submucous cellular tissue, we may have phlegmonous gastritis with submucous abscess. This is a rare but an undoubted consequence of alcoholism.

In some drinkers, chronic inflammation of the mucous membrane terminates in ulceration. *Ulcerous* gastritis is much rarer than simple chronic gastritis. The ulcerations are either one or several: they are situated principally at the points where we have seen that inflammation is most liable to occur. According to Dr. Lancereaux, the almost constant presence of the colouring matter of the blood at the bottom or round the edges of the ulcers, as well as the disposition which they have to be elongated in the course of the vessels, would seem to indicate that at least some of the ulcers are due to alteration or consecutive obliteration of these vessels. There would be necrosis of the mucous membrane from vascular obliteration.

In these cases, it is very evident that the drunkards are diseased persons, and suffer not only from anorexia, dyspepsia, and mucous vomiting, but have also acute pain in the stomach, a radically bad digestion, vomiting of food (sometimes incontrollable), and often gastrorrhagia.

The *liver* is the organ which comes next to the stomach in point of frequency of lesion. It is subject to two distinct kinds of alteration from the contact of alcoholic substances: it may either undergo fatty degeneration, or it may be the seat of chronic inflammation: in the former case, it is *steatosis;* and in the latter, *cirrhosis.*

Steatosis consists in the deposit of fat in the hepatic cells. This alteration of tissue is almost invariably met with in alcoholic drinkers. When partial, it is compatible with health—at least with apparent health. It appears to be then only the first stage of a more profound change, which is met with particularly in those who sink under the acute symptoms of alcoholism, such, for example, as *delirium tremens.* The liver is augmented in volume and pale in colour, where the lobules are infiltrated with fat.

When the steatosis is general, the liver presents a dull yellow hue, or is fawn-coloured: it has a granular or embossed appearance produced by the infiltrated and consequently enlarged lobules projecting beyond the surrounding tissue. The liver is more voluminous in this than in the preceding form of steatosis, and has sometimes its left more hypertrophied than its right lobe. The accumulation of fat, by distending the hepatic cells and acini, compresses the capillary vessels, and so, of necessity, induces anæmia of the parenchyma. Hence originates a new cause of paleness of the organ.

In these cases, there is no pain in the liver. By palpation and percussion, its increased volume can be ascertained. The stomach performs its work of digestion the more imperfectly that both it and the liver are diseased. Generally, the stomach is distended with gas, and tender when pressure is made on it. The stools are few in number, and have a pale argillaceous appearance. Diarrhœa occurs sometimes: hæmorrhages also take place, but not so frequently. Finally, Addison has pointed out a pale, waxy, cutaneous discoloration, in which the skin is supple, soft, and sometimes oily to the touch.

Cirrhosis "gin-drinker's liver," [*foie des buveurs de gin*] is characterised by an exuberant formation of interstitial cellular tissue. I shall tell you, in a special lecture on cirrhosis, that it is a disease not always identical with itself, and that the cause which produces it imparts to it a special form. In alcoholism, the liver is everywhere equally saturated with alcohol, and everywhere equally affected with disease. At first, there is increased volume and vascularity of the organ: at a later period, the interstitial exudation becomes trans-

formed into adventitious cellular tissue, which afterwards becomes fibrous, and then contracts: the organ everywhere decreases in bulk: the lobules and acini, indurated and diminished in size, slightly project above the depressions, more or less decided, which are everywhere constituted by the interlobular and inter-acinous partitions. The result is a general uneven state which is altogether characteristic. Here, I shall only mention—as I mean to return to the topic on a subsequent occasion—the profound alteration of nutrition which leads to progressive and altogether special emaciation, to possible hæmorrhages, and particularly to that kind of ascites which accompanies cirrhosis.

Along with this profound disorganisation of the liver, it is convenient to place transient, acute alterations of that organ, and particularly the functional disorders which follow alcoholic excesses. I refer to *jaundice*, which is apt to supervene some days after a debauch, and is preceded by more or less decided gastric disorder. It is probable that there is at first simple hyperæmia of the liver, caused by irritation from engorgement with alcoholic substances; but excesses are constantly repeated, and you can easily understand that the hyperæmia will also be reproduced, and will forthwith lead to an inflammatory condition, generally of a bastard character, the ultimate consequence of which will be—according to the nature of the case—steatosis or cirrhosis.

The *salivary glands* have been found by Dr. Lancereaux, to be "soft, yellowish, and having their epithelium manifestly invaded by granulo-adipose degeneration."

The *pancreas* has six times been seen by the same observer to be affected — sometimes altered in the same manner as the salivary glands, sometimes shrivelled and atrophied like the liver of cirrhosis. You see, therefore, that this organ undergoes changes similar to those of the liver, and also, that the mode in which they are produced is similar.

The *serous membranes of the abdomen*—the peritoneum, mesentery, and omentum—are the seat of an excess of fatty deposit, or of adhesive inflammation. At other times, as in a case observed by Dr. Blachez, the peritoneum was studded with ecchymotic spots produced by a mechanism which I shall immediately explain to you. Excessive fatty deposit in the omentum and mesentery, when coincident with the visceral lesions which I have pointed out, is sufficient to establish the existence of alcoholism. As to the adhesive neoplasm,

it is accompanied by a serous exudation, which is very scanty when the liver is fatty, or, in other words, when cirrhosis does not exist: [in cirrhosis, there is ascites]. The neoplastic exudation sometimes produces adhesions of the abdominal viscera. Besides this adhesive inflammation, Dr. Lancereaux has pointed out that there is a granular peritonitis peculiar to drunkards, which is characterised by small granular masses very similar to the tuberculous granulations of acute phthisis. "Along with these lesions," says Dr. Lancereaux, "there was fatty alteration of the liver, and in one case, an ulcer of the stomach. This occurred in robust persons in whose antecedents there was nothing tuberculous."

Were the *vena porta* more frequently examined in cases of alcoholism, it is exceedingly probable that it would be found to be very much altered, as it receives in the first instance the alcoholic substances proceeding from the stomach. Dr. Lancereaux has pointed out the occurrence of inflammation of the vena porta accompanied by pseudo-membranous exudation.

The *pulmonary artery* which receives the venous blood, and consequently the blood surcharged with alcohol coming by the vena cava from the liver, may, in drunkards, present a similar alteration. This, also, has been pointed out by Dr. Lancereaux. "There exists," remarks this able observer, "a form of arteritis which is characterised anatomically by membranous formations on the interior of the vessel. This form of arteritis which I have always met with in the pulmonary artery may determine to a great extent, and in a manner altogether mechanical, coagulation of the blood, leading to obstruction of the vessel and death. The frequency with which it is met with in drunkards does not seem to be fortuitous; and there is every reason to believe that it owes its origin to the abuse of alcoholic drinks."[1] One of the earliest symptoms is dyspnœa: it increases by slow degrees, and is a symptom the more remarkable that it is not indicated on auscultation by any physical sign, a circumstance which depends upon the cause of the dyspnœa not being seated in the respiratory passages, but in the pulmonary artery itself. There is either slight cyanosis, or there is loss of colour in the integuments. If the coagulation of blood be considerable, or if it be within a large trunk of the vessel, the thrombus inevitably causes rapid death from apnœa.

You can well understand that it is impossible for the lungs,

[1] Lancereaux:—*Gazette Médicale*, Paris, 1862.

through the medium of the pulmonary artery, to remain constantly in contact with the alcoholic substance, without their delicate tissue being injured. This is in reality what does occur. In drunkards, every variety of pulmonary lesion is met with, from congestion to inflammation and tubercle.

Pulmonary congestion is the most frequent of these lesions, for it is necessarily the first stage of all the others. It is generally located in the posterior margins and at the base of the lungs, as in cases where there exists adynamia. The pulmonary tissue is flaccid, soft, little aërated, but still capable of being inflated: it has a brownish colour, which is with difficulty removed by washing. One stage more—and there is true *hæmorrhagic infiltration:* the diseased vessels have burst. Finally, the pleura may be studded with ecchymotic spots. There is seldom an opportunity of observing these lesions, except when the patient sinks under the subacute symptoms, such as *delirium tremens.*

The symptoms are those of dyspnœa with a sensation of constriction in the chest, cough accompanied by mucous expectoration streaked with blood, disseminated crepitant and subcrepitant râles without bellows-murmur: in two words, the signs are those of congestion. The congestion is all the more natural, that a portion of the absorbed alcohol traverses the pulmonary tissue that it may be eliminated during expiration, and that in doing so, it must necessarily irritate that tissue.

Pneumonia, likewise, is the consequence of the lung being thus impregnated with alcohol. Not unfrequently, we find in the centre of the congested points, as I have just said, indurated lobules of a brownish, dirty yellow, or greenish colour, infiltrated with blood, pus, or fat. This is bastard pneumonia. There is also met with genuine pneumonia, usually adynamic or ataxo-adynamic, and frequently terminating in suppuration. It is sufficient to point out the frequency of pneumonia in drunkards: to give the signs by which to detect its presence is unnecessary. It is characterised by nervous complications, by the serious nature of the general symptoms, and by the possibility of the attack terminating in suppuration of the parenchyma. Another possible termination of the pneumonia of drunkards is its passing into the chronic state. Resolution does not take place, and the parenchyma becomes indurated. This has been pointed out by Magnus Huss.

Alcoholism debilitates: every affection which debilitates may lead to tuberculisation: independently of alcoholism, tuberculisation is

common: for all these reasons, it can be understood that under the influence of a predisposition or weakness on the one hand, and of constant irritation of the lung on the other, tubercle becomes developed. Bell of New York has refuted the strange fancy that the excessive use of alcoholic drinks protects from tuberculisation. He has shown that the reverse of this statement is the truth. My own experience leads me to the same conclusion. The pulmonary tuberculisation dependent on alcoholism may be either chronic or galloping; and that is all I have to say to you on the subject.

Hitherto, I have followed the alcohol in its passage from the stomach to the lungs: I have not traced it beyond the venous system or lesser circulation: I now propose to follow it through the greater circulation. Again, I say, that here it is less in quantity, part having been eliminated by the lungs; also, that it is more diluted, and consequently less irritating. However, it sometimes does not the less on that account determine direct lesions of the heart and arteries, particularly of the aorta.

Dr. Lancereaux says that the appearance of the heart is quite special. At first, it is scarcely larger than in the normal state; and is chiefly remarkable for the fatty deposit at its base and on the parietes. The fat does not merely line the heart: it likewise penetrates between the muscular fibres, and induces partial atrophy by the compression which it exerts upon them. Milky looking spots are observed on the surface. At a later period, the fleshy tissue of the heart is yellowish, soft, and very friable. At a still later date, the muscular fibres are altered, their striated appearance is less manifest, and they have either become granular or fatty. The myolemma is thick, and the cellular tissue, rendered exuberant by irritative congestion, ends by compressing and as it were partially suffocating the muscular tissue.

The endocardium may be affected by alcohol; but this class of lesions has not been well studied. Dr. Lancereaux found the aortic valves below the tubercles of Arantius thickened, white, greyish, and, to a slight extent, contracted or insufficient.

The lesions of the arterial system are a little better known than those of which I have been speaking. Magnus Huss pointed out the existence of atheromatous patches in the thoracic aorta and cerebral arteries of drinkers. In fact, throughout the entire arterial system, traces are to be found of the ravages committed by the passage and contact of alcohol. Dr. Lancereaux has met with—chiefly

in the thoracic aorta—patches, more or less thick, irregular in shape, and formed of cellular tissue. It is this which, in its ulterior metamorphoses, is transformed into an atheromatous patch in the large arteries, which is the starting point of fatty degeneration of the small arteries, and in particular of the small cerebral arteries where chiefly the lesion has been studied.

On 14th December, 1867, Dr. Blachez presented to the Medical Society of the Hospitals morbid parts in which were well seen, not only the lesions of which I now speak, but also the symptoms to which they might lead. Here, in a few words, is the case to which I refer:—

A man, 46 years of age, was admitted as a patient to the wards of Dr. Blachez. This patient was in a state of almost furious delirium which came in fits, during which he cried, vociferated, and endeavoured to strike every one near him. During intervals of comparative composure between the attacks, he looked around him with a stupid air. When touched, he cried out that he was being hurt. The entire cutaneous surface was in a state of hyperæsthesia. There was neither paralysis nor coma. The pulse was 120: the skin was covered with sweat.

On the following morning, during the visit, the patient was drowsy, but could be easily roused, though it was impossible to obtain from him any sign of intelligence: he had a fixed look, and talked incoherent nonsense. The hyperæsthesia continued, but without paralysis or contraction. Coma supervened during the morning, without a fit coming on, and without any fresh excitement. Before noon, the patient died.

At the autopsy, there was found "double meningeal hæmorrhage," to use the title under which Dr. Blachez has published his interesting case. In my opinion, however, the case was one of hemorrhagic meningitis, which, nosologically is a very different affair. In point of fact, at the convexity of each hemisphere, there was "a trembling gelatinous mass which elevated the dura mater." Upon cutting open the dura mater, a sanguineous effusion was found beneath it. The symptoms had at first been those of meningitis, and the patient had no coma till the last hours of life.

Besides other pathological changes, Dr. Blachez points out the atheromatous patches in the vessels at the base of the brain. Upon the thoracic pleura, there were numerous livid patches which were evidently ecchymotic. The lungs were exceedingly congested. The

internal surface of the pericardium was, like the pleura, covered with ecchymotic patches. Upon the heart, there were patches of a milky colour. The internal surface of the aorta, particularly at the arch, was as it were marbled with yellowish white stains, and in the same situation, the artery was thickened. On the peritoneum, there were ecchymotic patches. The liver was manifestly pale and hypertrophied. On examining it with the microscope, it was found to be less fatty than might otherwise have been supposed. All the hepatic cells could be easily recognised, but many of them contained more than the normal quantity of fat, and some fine fatty granulations were scattered around them. The volume of the kidneys was enormous: this increase in their size resulted from hypertrophy of the cortical substance, which had a greyish colour, a greasy aspect, and was studded with small ecchymoses. The cortical tubes were infiltrated with a protean, finely granular substance.

Dr. Blachez asks whether the numerous hemorrhages observed in this patient were not dependent upon an alteration in the blood having been induced by alcohol.[1] But, concurring with Dr. Peter, I believe that the reality of this alteration in the blood has not yet been demonstrated; and consequently, I prefer to explain all the phenomena by the positive lesions of the vessels. I entirely concur in Dr. Peter's view, that it is a magnificent example of alcoholism in its acute and rapidly disorganising form. It is probable, that the individual was in the habit of daily imbibing large quantities of alcoholic substances, and that he took more brandy than wine.

"Therefore," remarks Dr. Peter, "it is certain that this man had general inflammation of the arterial system. As a necessary consequence of this general inflammation, the blood must have carried everywhere an irritating substance; and to enable this irritating substance to act so rapidly, and with so much intensity, it must have existed very abundantly in the blood. Now we know, that the irritating substance which habitually produces similar disorders is diluted alcohol.

"Under such circumstances, there is evidently inflammation of the arterial system; and it was this inflammation which caused all the disasters, sometimes exciting inflammation and sometimes inducing hemorrhage. The irritant poison circulating from artery to arterial branch, and from arterial branch to capillary, produced everywhere

[1] Blachez:—The *Union Médicale* for April and May, 1867.

similar primary disorders, and consecutive lesions, varying according to the diameter or resisting power of the vessel. In the aorta, the white patches are the result of the proliferation of nuclei, the first stage of an irritative process.

"In the kidneys, the ecchymotic spots, disseminated in the form of red spherical points, are apparently hemorrhages in the situation of the Malpighian tufts, that is to say, the minute tortuous arteries are ruptured where they curve, in consequence of the pathological change induced in them by alcoholic irritation.

"That is not all: in addition to the primary vascular lesion, there is a consecutive visceral lesion.

"In the patient of Dr. Blachez, the liver had undergone a morbid change: it was not only increased in volume, but was likewise pale in colour, considerably infiltrated with fat, while at the same time, it was the seat of a proliferation of nuclei of adventitious tissue.

"There was also a morbid state of the kidney, which, like the liver, was pale, and like it was the seat of a proliferation of nuclei of epithelium in the tubular substance, irrespective, I repeat, of the hemorrhages in the situation of the tufts.

"It is probable that similar lesions, if looked for, would have been found in other glands, such as the pancreas, salivary glands, and testicles.

"It is to be regretted," continues Dr. Peter, "that no inquiry was made into the state of the portal vein and pulmonary artery, the vessels which first come in contact with the absorbed alcohol. Had they been examined, there would probably have been found inflammation of their internal membrane, which would have imparted a great degree of certainty to the etiology and pathogeny which I propose.

"To sum up:—these numerous lesions—and they might have been found to be still more numerous—seem to me to have been due to one and the same cause, irritation produced by the contact of an alterative substance in the blood, that substance being alcohol.

"If from a purely micographic point of view, the white patches on the aorta are due to a proliferation of nuclei, this proliferation (the primary consequence of the irritative process of Virchow) looked at from a nosological point of view, is only the first stage of inflammation. These nuclei would afterwards have been transformed into fat by retrogression, and one would have had *atheroma*, which would subsequently have become a calcareous plate, by a deposit of salts taking the place of fatty molecules.

"Also, had the patient lived long enough, similar changes would have been observed in the liver and kidneys: there would have been found the fatty liver and Brightian kidneys of drunkards [*reins brightiques des ivrognes*].

"The pathological drama was suddenly stopped at the first act, by death, the result of pachymeningitic hemorrhage.

"This abrupt termination was a fortunate occurrence, if looked at from a scientific point of view, as it enabled us to detect the first phase of a series of lesions which generally we do not observe except in the more advanced stages—that is, in fatty degeneration." [1]

I have quoted *in extenso* these remarks of Dr. Peter, because they connect by a common etiological chain all the lesions observed in alcoholism, and because they express a pathogenic theory which I accept.

It appears then that the patient of Dr. Blachez presented a combination of the lesions of acute and chronic alcoholism. The alcoholic impregnation of the brain caused meningitis, and the morbid change in the vessels led to hemorrhagic meningitis. When the alcoholic impregnation has been great, *delirium tremens* is the result: it causes by its action on the nervous system, in respect of the motor powers, tremors—in respect of sensation, a great variety of affections—and in respect of the mind, dullness, alienation, or dementia.

Were I to discuss at greater length the lesions due to alcoholism, I should unduly extend these lectures: my chief aim has been to show you that alcohol exercises a similar action upon every part of the organism.

There is one organ, however, of which I wish to speak before I conclude: that organ is the *kidney*. It may be affected either with *granular* or *fatty degeneration*. In the former case, the morbid change is chiefly in the cellular tissue of the organ, and is analagous in its nature to cirrhosis of the liver: in the second instance, it is principally the epithelium which is infiltrated with fatty matter. In the former, the kidney gradually diminishes in size, and acquires an unequal surface from contraction of the cellular tissue: the volume of the cortical substance becomes less and less, in consequence of the atrophy occasioned by the thickening of the cellulo-fibrous tissue, particularly in the situation of the tufts. Finally, the epithelium is

[1] Peter (Michel):—*Bulletin de la Société Médicale des Hôpitaux* for December, 1866.

granular and is frequently altered. In fatty degeneration, the kidney remains bulky, and retains the normal smoothness of surface; but it gradually loses its colour, and becomes yellowish, either wholly or in isolated patches. The tubuli become more voluminous and infiltrated with granulations (fatty for the most part), developed in the epithelial cells, which are turgid and deformed. In both cases, there is Bright's disease, accompanied by symptoms with which you are acquainted. It is impossible to avoid looking upon these lesions of the liver and kidney as similar in their nature, and as originating in the same cause, that cause being the action of alcohol.

Again, it is this very same action, which, by dilating the vessels of the *skin*, produces lividity of the face, particularly of the nose, where the circulation being naturally more sluggish, the blood circulates still less actively in consequence of the vascular dilatation. Similar changes take place in the glands of the skin as in the other glands: the sudiparous and sebaceous glands generally undergo the granulo-fatty alteration: in point of fact, we generally find the epithelium of the sudiparous glands in a granular state, and the sebaceous glands filled with an enormous quantity of fatty matter. Inflammation is another process by which the sebaceous glands are altered: it is this which constitutes the *acne rosacea* (sometimes copper-coloured)—the indelible stigma of drunkenness.

Let me add in conclusion, that all these lesions are not met with at the same time in the same individual; and that the most common are the lesions of the stomach and liver, after which come those of the nervous centres, and finally those of the kidneys.[1]

Gentlemen, I have endeavoured in this rapid sketch to place before you the manner in which the tissues of the human body are acted upon by alcohol taken to excess. To enable me to do so, I have had to follow it stage by stage in its passage through the organism, and to show you that everywhere it is equally a destroyer; and also, that it everywhere produces lesions essentially similar, presenting only differences in appearance arising from the difference of tissue of the affected organs. It is the aggregate of these lesions, and of the symptoms which correspond to them, which constitute

[1] See the following works:—Racle (A.)—De l'Alcoolisme: *thèse d'agrégation*, 1860.—Fournier (Alfred)—Article Alcoolisme in the *Dictionnaire de Médecine et de Chirurgie Pratiques, T. I: Paris*, 1864.—and particularly the excellent Article, Alcoolisme by Dr. Lancereaux in the *Dictionnaire Encyclopédique des Sciences Médicales*, 1865.

true alcoholism in its acute or chronic form. Whether alcoholism be acute or chronic, it is always the same organs which are injured; and the symptoms observed, though they vary in degree, are always the same in kind. Thus, to name together *delirium tremens*, gastric symptoms, jaundice, and perhaps also disturbance of the urinary secretion, is to give a succinct description of acute alcoholism: alcoholic tremors, general dulness of the nervous system, chronic or ulcerous gastritis, cirrhosis, and Bright's disease are the phenomena of inveterate alcoholism. In three words, the difference presented by the latter may be described as consisting in prolongation of the cause, greater intensity in the effects, and more profound morbid alteration of organs.

LECTURE LXII.

SPERMATORRHŒA.

Local Phenomena.—General Symptoms.—Symptoms which may be mistaken for manifestations of very different diseases from those to which they belong.—Spermatorrhœa depends on Different Causes.—Spermatorrhœa Consequent upon Chronic Irritation of the Urinary Passages and Rectum.—Spermatorrhœa from Excessive Contractility of the Vesiculæ Seminales.—Spermatorrhœa from Atony of the Ejaculatory Canals.—Treatment must vary according to the Nature of the Cause.—Treatment of the two last mentioned forms by Compression and the Topical Application of Heat and Cold according to the Indications.

Gentlemen:—By *involuntary seminal emissions* or *Spermatorrhœa*, we understand those losses or evacuations of seminal fluid which either take place without any, or with inadequate erotic excitement. In the normal state of a properly constituted person, the emission of seminal fluid requires not only that the venereal orgasm should be carried to a very high degree, but also that a series of acts be repeated for a longer or shorter time: there is required the mechanical act of copulation, or the use of some other analogous means. Amorous desires, be they ever so keen, occurring even in the strongest and most continent persons, do not in general cause spontaneous ejaculation of semen: nor is ejaculation brought about by mere contact with the object of desire. When ejaculation occurs independent of the erotic excitement generally required, there is an involuntary loss of semen. Bear in mind, however, that, using the term in a wide acceptation, I for the moment place in one general group very different degrees of the affection, beginning with nocturnal pollution properly so called, an occurrence which in many cases is not morbid, and culminating in spermatorrhœa, the malady upon which I propose now to address you.

The pollutions which spontaneously occur in persons who are too continent, supervene during sleep under the influence of lascivious dreams accompanied by erection and a high degree of venereal orgasm, do not generally come within the province of the physician, inasmuch as they generally indicate an excess of health and vigour, rather than a state of feebleness and disease. The individuals who have these emissions usually experience on awaking a general feeling of comfort succeeding to the uneasy sensations by which they had often been previously tormented: they feel themselves freer, more buoyant in spirits and more at their ease: they are in the position of persons who have satisfied a physical want. I ought, however, to remark, that nocturnal pollutions in chaste persons in good health occur much less frequently than is commonly believed. A very vigorous man may entirely abstain from women for months without having nocturnal pollutions; and, speaking generally, they ought to be of rare occurrence, excepting perhaps during the first years of manhood. Should they take place every month, still more, should they occur every fortnight, or every week, should the loss of semen happen in this way even less frequently than in those who resort in moderation to coitus, there will nevertheless be some bad effect produced: and although in the morning after the pollution, there is a feeling of well-being, the individual is on a decline which may lead to bad health. In fact, the seminal emission, under the influence of different causes, and even from mere habit, soon becomes a pathological occurrence: it recurs at intervals less and less distant, it ceases to be active and becomes passive, that is to say, happens without dreams, without erection, and without any erotic sensations being experienced. Ere long, the individual will cease to be conscious of what took place during the night, and would remain quite unaware of the occurrence, were it not for the visible traces of the semen. These are the pollutions which constitute the first degree of the malady we are now studying.

Almost always, if not always, spermatorrhœa properly so called begins with nocturnal pollutions. They have in the first instance been the result of erotic dreams: they recur frequently: through habit, the frequency increases to such an extent that they take place not only once every night, but several times in the same night. At a more advanced stage of the disease, the seminal emission occurs without erethism, and also without erection, by which, in the first instance, it used to be preceded and accompanied. At all events,

the patients are quite unconscious of having experienced any voluptuous sensations, and it is only on awaking that they become aware of what has happened to them when asleep.

Then also, in place of experiencing a feeling of well-being, they feel out of condition and weary: they complain of heaviness of the head, and a certain amount of mental wandering and incoherency of ideas: they are not in a fit state to work either with mind or body.

After the lapse of a certain time, there are diurnal as well as nocturnal pollutions. At first, there is still the necessity of a certain degree of orgasm, but an imperfect erection lasting only for a short duration will suffice to cause emission. If the patients copulate, ejaculation takes place immediately: it sometimes happens that the introduction of the penis has hardly began, when the venereal act ends, and the erection suddenly ceases. At a later period, still less will be required: mere touching or rubbing, such as occurs in riding on horseback, or in the movements of a carriage or swing, and at other times, the mere sight of objects suggestive of lascivious ideas will be the immediate determining causes of a greater or less flow of semen.

Under all circumstances, even in those cases in which the nocturnal pollutions are compatible with perfect health, involuntary seminal emissions take place under the influence of a sort of erotic excitement, but an inadequate erotic excitement, as compared to that which there ought to be, were the ejaculation accomplished in a normal manner. But when the spermatorrhœa reaches an advanced stage, the seminal emissions take place without there having been the least previous excitement.

This kind of seminal emission may occur, however, irrespective of the disease of which I am now speaking, but it is then only a transient and unimportant symptom. For example, an individual, generally in good health, becomes affected with obstinate constipation. When at stool, he has a seminal emission: it is an unimportant mechanical phenomenon; for in this case, the seminal emission is the result of pressure upon the seminal vesicles by the excrement which the energetic intestinal contractions are expelling.

This is a fact which we do not require to stop to consider: but it is otherwise in respect of spermatorrhœal patients having habitual seminal emissions not only in the act of defecation, but also during micturition. In the first referred to class of cases, the seminal flux

only takes place in small quantity, and under the influence of a violent effort, while in the latter, it supervenes when there is no straining—when the motions are diarrhœal—as well as when they are hard and solid.

The emission of urine will excite the emission of semen, which will come sometimes with the first, but more generally with the last portion of urine. Lallemand (of Montpellier), to whom, as you know, we are indebted for the most complete inquiry which has been made into this subject, says that seminal emissions supervening during micturition are the most serious in their character, and the most rebellious under treatment: they are also the most difficult to recognise, in consequence of the change produced on the semen by its mixture with the urine.[1]

Lallemand, however, has pointed out certain physical and microscopical characters which may be of use in the diagnosis of such cases. Sometimes, the patients themselves perceive that there is a change in the appearance of their urine: they observe that the last drops passed are thick, glutinous, and viscid, liable to form small curdled clots, which stopping at the entrance of the meatus, acquire a sticky starchy consistence, and leave a mark on the linen like that caused by the starch used in the laundry.

If the urine in the vessel is examined as soon as it is passed, small bodies may be seen rolling at the bottom of the fluid which are variable in size, semi-transparent, irregularly spherical, and in appearance very much resembling grains of semolina. These bodies are soft, and do not adhere to the sides of the vessel: they appear in the urine before it has cooled, and in urine which is quite clear—a circumstance which prevents the semen being mistaken for a deposit of urinary salts.

When the malady has advanced still farther, the characters now indicated are absent. The urine does not deposit granular bodies sufficiently large to collect at the bottom of the vessel, but it contains a thick, homogeneous, yellowish cloudiness studded with small brilliant points occupying the inferior strata, and resembles the deposit which forms in a concentrated decoction of barley or rice. Lallemand says that the presence in the urine of these granular bodies leaves no doubt as to the nature of the cloudiness in which

[1] Lallemand:—Des Pertes Séminales Involuntaires. Paris, 1836—1842. Three volumes, 8vo: published in five parts.

they are observed. Certain precautions have to be taken to enable us to lay hold of the characteristic appearance of which I am now speaking. Urine passed at different periods of the day does not always present the same appearance, so that what is passed at each micturition ought to be kept in a separate vessel. Generally speaking, the urine voided in the morning, particularly when the patient has passed a bad night, are the most loaded: at other times, the urine most loaded is that voided after venereal excitement, violent mental emotions, or when digestion has been difficult. Sudden chilling of the body may produce the same effects. During the day, the urine is generally clear.

By the assistance of the microscope, Lallemand ascertained that the cloudiness of which I have been speaking is in a great measure due to the presence of semen mixed with the secretion from the mucous membrane of the urinary passages, and that the shining points in the cloudiness consist of the matter excreted by the seminal vesicles. A fact still more interesting has been elicited by microscopical inquiry, viz., the altered state of the spermatozoa in patients affected with spermatorrhœa. The animalcules decrease in number and volume, and, in very severe cases, assume a spherical form: moreover, their vitality diminishes as the disease advances.

Gentlemen, I have told you that the patients themselves sometimes perceive a physical change in the character of the urine: I would now add that some of them complain of experiencing at the time of micturition certain sensations which announce the occurrence of the pollutions. There is felt a peculiar grazing sensation caused by the passage of the urine, and arising from its unusual density: there are spasmodic contractions: there is pain extending from the neck of the bladder to the glans penis and margin of the anus: there is also a shiver accompanied by a general feeling of discomfort. Lallemand states that those who are accustomed to these peculiar coincidences know that they will find in their urine a flocculent deposit containing the granulations of which I have been speaking. So complete is their conviction on this point, that they immediately experience a cold sweat accompanied by faintness.

Irrespective of the changes in the urine due to the presence of semen, there are also others which are pretty frequently met with. They are connected with certain complications, which often accompany spermatorrhœa. These complications are acute and chronic cystitis; inflammatory affections of the prostate gland, ejaculatory

canals, and even of the seminal vesicles—inflammations leading to the formation of morbid mucous secretions, which explain the presence of mucus, purulent mucosity, and pure pus, all contributing to produce the cloudiness of which I have spoken.

These coincident inflammations also explain a certain number of the symptoms complained of by the patients, such as discomfort and weight in the hypogastric, perineal, and anal regions—pain accompanying any slight over-exertion, walking, and riding, which are acutely felt even after sitting a long time.

We have now reached the question:—What are the symptoms which usually bring in their train involuntary seminal emissions?

Lallemand, though he possibly exaggerated the disastrous consequences of this disease, is certainly entitled to the merit of having in a special manner called the attention of the profession to this subject; and no one more than the illustrious physician of Montpellier has diffused information on this point, so important both in the science and art of medicine. He thought, and correctly thought, as you will see immediately, that spermatorrhœa was the cause of certain diseases of the nervous system and affections of the moral and intellectual powers [*de vésanies*]; but unquestionably he exaggerated, attributing to involuntary seminal emissions some affections not in any way originating in this cause. He has not sufficiently recognised the fact, that spermatorrhœa is not necessarily the cause of the different neuroses described in his book, but is in many cases only the expression of a nervous disorder which first shows itself as spermatorrhœa, and then assumes forms of a much more serious character.

Before entering into this part of our subject, let us recall what takes place after the act of copulation. Coitus is succeeded by a state of depression and weariness: if inordinately repeated, there is a still greater state of bodily prostration, the intellectual powers are blunted, and the mind temporarily loses its accustomed energy. Cessation of erection is, however, the immediate result of the accomplishment of the venereal act. Whenever ejaculation has taken place, erection ceases more or less quickly in most animals as well as in man: a certain time is required to recruit the strength; and to restore the erection, new excitement is required. In a word, frigidity [*frigidité*] is the immediate result of ejaculation, a frigidity, of course which is relative, and, under normal conditions, temporary. Thus it becomes easy to understand how it is that involuntary

seminal emissions recurring at short intervals have a principal tendency to lead to absolute frigidity, and finally to impotence.

Fully understand, Gentlemen, that I attribute this impotence to the state of feebleness which is associated with habitual loss of semen, and not as do many physicians to the shaking of the nervous system accompanying the venereal act. In support of my opinion, allow me to enter into some explanatory details.

It has been alleged, I said, that the feebleness and prostration which succeed ejaculation, or the last stage of the venereal act, to speak more correctly, depended upon the shaking of the nervous system, experienced during the accomplishment of that act. In my opinion, however, that is an element which only to a small extent enters into the causation of the exhaustion.

Reflect on what takes place in the woman. In her, the excitement of the nervous system, what is called the cynic spasm, is quite as energetic as in the man: often, it is even much stronger, and nevertheless a woman can, generally speaking, engage in copulation, and repeatedly accomplish a complete venereal act within a very short space of time, at much shorter intervals than a man could, and yet without her experiencing extreme fatigue or much exhaustion of strength. It follows, therefore that the cynic spasm, the shaking of the nervous system, which accompanies coitus cannot be regarded as the principal cause of the debility and exhausted state induced by it; and consequently, cannot be looked upon as the principal cause of the frigidity and impotence, which must be attributed to the loss of seminal fluid. If we turn to pathological data, we shall there again find the proof we are in quest of; for the debility consecutive upon passive pollutions, that is to say, pollutions irrespective of erotic dreams, without erections and without voluptuous sensations, is much greater than when the pollutions have been active and accompanied by a certain degree of cynic spasm.

Be that as it may, impotence is one of the first consequences of involuntary seminal emissions—I say one of the first consequences, as the malady need not have long existed for this symptom to show itself.

Loss of procreative power is the usual result. This condition, however, must not be confounded with impotence. An impotent man may be fit to generate, while a man in the full vigour of virile power may be unfruitful. The infecundity of the latter may depend on different causes. Thus, with every appearance of virility, the

man may be destitute of procreative power, because his semen has not the necessary qualities, is destitute of spermatozoa, or if it do contain these little animalcules, they are altered and misshapen. Men in whom the testicles have not descended into the scrotum, are unfruitful, but not impotent. Cryptorchidous men and mongrel animals, while they are perfectly capable of discharging into the female, and are even exceedingly lascivious, are unfruitful. Infecundity in the man may also depend upon imperfection or morbid alteration of the external genital organs. The penis may be deficient in length, either by natural conformation, or by having been accidentally shortened, as, for example, when it is, so to speak, obliterated by a tumour in its vicinity, a hydrocele, or a scrotal hernia; on the other hand, it may be excessive in length or thickness, or its direction may be vicious; again, there may be epispadias, hypospadias, or phymosis—anomalous conformations which may prevent the ejaculated semen from being deposited in the manner in which it ought in the sexual organs of the woman: once more, stricture of the urethra may be a cause of infecundity by embarrassing the passage of the semen. Cases are on record in which stricture was produced during coitus by the erection being too vigorous, so causing the semen to regurgitate into the bladder, or preventing its being ejaculated till the turgescence of the penis had ceased.

Infecundity, therefore, does not necessarily imply impotence; and as I have said, the latter may exist—only of course to a limited extent—without the individual having lost his procreative power. To enable such a person to engender, it would only be necessary to introduce the penis completely within the vulvar passage, for then, even without erection, fecundation might take place.

In the subjects of spermatorrhœa, infecundity may depend upon ejaculation occurring before introduction of the penis into the sexual parts of the woman; or it may arise, notwithstanding introduction, from the ejaculation being too feeble to send the semen far enough: in both these cases, the uterus may not be sufficiently excited. The principal cause, however, of infecundity in spermatorrhœa is the alteration of the semen, which either does not contain spermatozoa, or only contains spermatozoa which are malformed or essentially changed in their nature.

Impotence and infecundity are far from being the only effects of involuntary seminal emissions; they may also originate various nervous diseases. Under this class of affections may be placed various dis-

turbances of the great functions of organic life, the origin of which is often misunderstood.

During the early period of the disease, the appetite may not only remain, but may even be increased; but the sensation of hunger is not that of ordinary hunger: there are twinging pains, a feeling of discomfort, and a sinking sensation temporarily relieved by taking a small quantity of food; soon, however, a loathing of food sets in, and to satisfy nature's demand for nourishment, the patients endeavour to stimulate the appetite by strongly spiced meats and exciting drinks. The results of this kind of feeding are irritation of the stomach and difficult digestion. The exciting food and surcharged state of the alimentary canal produce a notable increase in the involuntary emissions. Gastric and intestinal symptoms, varying according to the individual, and varying from day to day in the same individual, set in: constipation is succeeded by diarrhœa, which finally establishes itself in a chronic form, becoming a permanent and direct cause of spermatorrhœa.

The exhaustion and general debility produced by habitually involuntary loss of semen necessarily increase under the influence of disturbed nutritive functions. The victim of spermatorrhœa falls into a state of extreme wasting. He loses colour, the complexion becomes pale, and the skin acquires a yellowish leaden hue: his eyes become encircled with a blue ring, hollow, dull, and expressionless. He is easily injured by reduction of the external temperature; and he progressively loses his moral and physical energy. His muscular activity is more and more impaired: he becomes unable to sustain bodily exercise, even for a short time, without complaining of panting and dyspnœa; and in proportion to the advance of the disease, so increases the difficulty of performing muscular movements. It is an extraordinary fact, and one which according to Lallemand is a pathognomonic phenomenon of spermatorrhœa, that in conjunction with this feebleness—even when it exists to an extreme degree—the patient has an inconquerable desire to move, and, even when hardly able to stir, is impelled by physical restlessness to seek constantly to go from place to place.

Palpitation of the heart, and an accelerated, small, feeble pulse give evidence of disordered sanguinification, and anæmia is not unfrequently manifested by a vascular bellows-murmur. I have referred to the shortness of breath complained of by the patients on taking slightly prolonged exercise; at a later period, this oppressed

breathing is constant, rest not causing it to cease: the respirations are slowly drawn, few in number, and not deep. Certain patients are tormented by a constant dry cough, and suffer from neuralgic pains in some part of the chest: auscultation reveals a feebleness of the respiratory murmur depending upon general debility.

The sensorial functions are variously modified. A form of anæsthesia exists, which from its mobility may be compared to that observed in hysterical and hypochondriacal patients: sometimes in the hands, sometimes in the chest, sometimes in the abdomen, and sometimes in the integument of other parts of the body, the tactile sensibility is obtuse in a surface more or less extensive, and for a longer or shorter period. The individuals complain of very transitory sensations of heat, burning, or cold: they compare them to sensations caused by a current of electricity, by cold air, or by tepid water; they also complain of pains similar to those produced by a violent squeeze, by a contusion, or by tingling, of which the back and shins are the principal seat.

At last, the special senses participate in the general disorder of the system. *Disorders of the sense of sight* arise. Complete amaurosis may occur, beginning with amblyopia or diplopia, though cases of this description are rare. The impaired vision is accompanied by extreme sensibility to light, and a more or less remarkable dilatation of the pupils. The *sense of hearing* loses its delicacy and precision: it becomes exceedingly sensitive; there is buzzing, ringing, and singing in the ears, symptoms which sometimes proceed to such a length as to constitute complete deafness. The *senses of taste and smell* may also be perverted.

Pains in the head and vertigo, symptoms which constitute part of the concomitant train of phenomena of spermatorrhœa, are most palpable when the patients have difficult digestion, when they have attempted somewhat sustained mental exertion, or when they have passed sleepless nights. Their sleep is generally light, and but little restorative: as the involuntary seminal emissions take place most frequently during the night, they are more exhausted than before they fell asleep. At an advanced stage of the disease, there may be complete insomnia: when it is so, the patients pass the night in a state of great excitement, covering and uncovering themselves, getting up and lying down, changing their position every moment without ever finding one more convenient. When sleep does come at last, it is troubled by painful night-mare. These

distressing nights leave behind them extreme fatigue: and during the whole day, the patients remain in a sort of brutish stupidity of which they are conscious, a circumstance which explains the mental depression, hopelessness, and melancholy which make them seek to fly from every kind of society.

The patients undergo a great moral change. Wholly engrossed with their own state of health, they are indifferent to the circumstances by which those around them may be affected: they are exceedingly pusillanimous, irascible, and as insupportable to others as to themselves. Their memory becomes weak; and this enfeebling of the memory, combined with a certain degree of paralysis of the tongue, combined also with feebleness of voice and hesitating speech, makes it difficult for them to express their ideas, the elaboration of which, moreover, is less active and less precise.

Finally, the disturbance of the intellectual faculties may proceed to such an extent as to constitute insanity. The insanity may be temporary, and, remaining entirely subordinate to its cause, may be recovered from, when recovery from the spermatorrhœa takes place: but it may also be persistent, continuing long after the complete cessation of the spermatorrhœa which caused its evolution. Lallemand clearly indicated this capital fact, and observed that the most common forms of insanity which occur as consequences of spermatorrhœa are hypochondriasis, melancholia, and lypomania, complicated sometimes with a tendency to commit suicide.

This long programme of symptoms, upon which Lallemand has dilated with so much care—with perhaps too much care—recalls the characteristic features of confirmed hypochondriasis.

In general, it is not usual to find a young man suffering from severe hypochondriasis who has not spermatorrhœa. There are, however, numerous cases of hypochondriasis occurring irrespective of involuntary seminal emissions. But before I enter upon the doctrinal question touching the relation of spermatorrhœa to the state of the nervous system, I wish to bespeak your attention to certain material causes of spermatorrhœa.

It has been observed that spermatorrhœa is rather common among persons who have an unusually long prepuce. In such cases, the sebaceous secretion accumulates around the gland, which it irritates; and it can be understood that, when these conditions exist in persons predisposed to spermatorrhœa, ejaculations may occur as readily as they occur in the same persons when they have the least contact with

a woman. Lallemand has frequently seen, that in such cases circumcision, if it be not a means of cure, has at least an extraordinary influence in diminishing the extent of the spermatorrhœa.

But disproportionate length of the prepuce has relation to another cause which did not escape the sagacity of Lallemand—I refer to the imperfect development of the corpora cavernosa. In this case, we must look to the nervous system for the original cause of the affection. Individuals affected in this way are the children of parents nearly related by blood to each other, of insane and epileptic persons; or at least some members of their family are insane, epileptic, or the subjects of hare-lip. They have a pre-existing lesion of the nervous system, whence follows a complete series of consequences, and first of all an imperfect evolution of certain organs—on the other hand, a constant tendency to neuroses in general, and to spermatorrhœa in particular, the latter being in reality a neurosis of the organs of generation.

A similar explanation may be given of the pollutions which occur in monorchidous and cryptorchidous persons, and in individuals affected with hypospadias or epispadias: in these subjects, there exist both arrested development of the organ, and disorder of its function consequent upon a bad congenital condition of the nervous system: the congenital material lesion, as well as the disorder of the function with which the organ is charged, express one and the same thing, that is to say, a vice in organic evolution.

To return to the very various nervous symptoms which I have already enumerated in detail, let me remark, that you will meet with those which signalize the beginning of locomotar ataxy such as diplopia, amblyopia, the darting pains in the limbs; and let me remind you that, as a corollary, I have told you that locomotar ataxy is often preceded by seminal emissions.

Gentlemen, I have already stated my opinion regarding the extraordinary influence which seminal losses have upon the entire economy, but particularly upon the nervous system. I am convinced that Lallemand has shaded his picture much too darkly, and that he has made a great mistake in attributing perturbations of the nervous system to exhaustion caused by excessive and too frequently repeated discharge of semen, when the *nervous disorders* might more justly be regarded as the *cause* of the spermatorrhœa.

Permit me, Gentlemen, to expatiate a little upon this opinion. First of all, we know by experience, that a large proportion of the

young men who have spermatorrhœa have had *nocturnal incontinence of urine* in infancy: this is a very serious nervous symptom, and it often happens that the same subjects show eccentricities of character, irritability, and unequivocal signs of hypochondria at an age when hypochondria is very rare. Could we penetrate into family secrets, we should often find that among the predecessors of our patients there were brothers or sisters who had had serious diseases of the nervous system, such as hypochondria, insanity, epilepsy, and locomotar ataxy. We should thus find as an explanation of the spermatorrhœa, as an explanation of the nocturnal incontinence of urine, hereditary and personal predisposing causes; and should, therefore, have no right to attribute the nervous symptoms to the spermatorrhœa. It is far more reasonable to think and say that the nocturnal incontinence of urine and the spermatorrhœa are consequences of an unhealthy state of the encephalon, and particularly of the spinal marrow, a state the nature of which it is not easy to specify.

To illustrate this point, let me give you some examples which occurred within a short period in my private practice.

On 10th April, 1866, I was consulted by M. C— who had married his cousin german. He had by this marriage two sons, whom I have seen several times, The eldest, 9 years of age, is a delicate boy, and affected with nocturnal incontinence of urine: the youngest, eight years of age, has from his birth been an epileptic idiot.

On 13th April, I was consulted in the case of a young lady of 19, who was beautiful and healthy looking, but had been an epileptic for two years. She had not had nocturnal incontinence of urine in childhood, but her father had suffered from it till he was 7 years old.

On 16th April, I was consulted by a lady of 34 years of age: she had had puerperal mania. Of her two daughters, one was affected with nocturnal incontinence of urine till she was 12 years old: the other has hysterical paralysis. Father and mother are first cousins.

On 19th April, I was consulted on behalf of two young men whose father died insane: one was 24, and the other 21 years of age: both had frequent seminal emissions, and both led very loose lives.

On 20th April, I was consulted by a gentleman who had an insane brother. He himself had never had nocturnal incontinence of urine,

but had long suffered from very abundant and very frequent spermatorrhœa. For two years, he has had hypochondria almost proceeding to nosomania.

On 3rd May, I was consulted by a young man of 21, who had had incontinence of urine till he was 14 years old. From infancy, he had been a sufferer from epilepsy, and had had both the "grand mal" and the "petit mal." I was told that nervous diseases had never shown themselves in the family.

On 10th May, I received in my consulting room a young lady from Copenhagen, who up to the age of 5 years had had nocturnal incontinence of urine. From that time, she had had epileptic attacks particularly in the vertiginous form.

On the same day, I saw at Auteuil, at the house of M. Beni-Barde, a gentleman aged 30: his mother was very nervous and his grandmother had been insane: he himself had had nocturnal incontinence of urine till he was 12 years old: from 16 onwards till now, he has had spermatorrhœa: at present, he has hypochondriacal symptoms, the most curious nervous disorders, and apparently there is also imminent insanity.

On 11th May, I saw a young lady of Mezidon, aged 20. Her father was a lunatic, and her grandmother an hysterical subject. Till the age of 12, she had suffered from nocturnal incontinence of urine, and subsequently from epilepsy.

On 21st May, I was consulted in the case of a young lady of 13, the child of parents who were nearly related by blood. Till she was 5 or 6 years old, she had nocturnal incontinence of urine: from that date she had had vertiginous epilepsy, and sometimes the "grand mal."

On 22nd May, I was consulted by a young man, aged 22, who had nocturnal incontinence of urine till he was 6 or 7 years old. From the age of puberty onwards, he had had spermatorrhœa. A brother was paraplegic.

On 26th May, a gentleman of 45 consulted me. For two years he had had epilepsy. His son, 14 years of age, had nocturnal incontinence of urine.

On 3rd July, a Spaniard, aged 34, came to consult me. Till he was 10 years old, he had had incontinence of urine: from that age and onwards, he had had spermatorrhœa: he is now impotent: for the last three months, he has been subject to epileptic attacks.

On 5th July, I saw a young man of 22, who had suffered from

ncontinence of urine and fæces since he was 9 years of age. From puberty onwards, he had had spermatorrhœa. His mother and a brother were epileptics.

On 9th October, I saw an epileptic young man of eighteen. He had not had nocturnal incontinence of urine in childhood: but he had nocturnal spermatorrhœa about three times a week. The symptoms were not hereditary.

On 11th October, I saw a young man, a twin, of 25 years of age. In childhood, he had had incontinence of urine: he was when I saw him, suffering from nocturnal and diurnal spermatorrhœa. He felt much more tired after sexual intercourse than after a nocturnal pollution. He was the subject of very decided anaphrodisia, and the seminal emission took place almost immediately.

I do not lengthen this catalogue of cases, though, without any difficulty, I could largely do so. You will perceive that the relatively considerable number of almost identical facts occurring within a very limited space of time is sufficient to prove, in the first instance, the relation between the genito-urinary and nervous symptoms of an individual, and also the influence on children of the consanguinity of their parents, and of the state of their nervous system; then, on the other hand, it establishes the existence of a chain of morbid phenomena, almost always the same in character, which begins with nocturnal incontinence of urine, continues with spermatorrhœa, and culminates in hypochondria, epilepsy, and insanity. This shows you that all the symptoms originate in one and the same cause, and that that cause is a primary morbid cause pertaining to the nervous system.

There is yet another consideration to which I wish to direct your attention. I am frequently consulted by men who have seminal emissions, and also suffer from the series of nervous symptoms of which Lallemand has so complaisantly drawn the picture. Upon entering into details, it is found that the nocturnal pollutions do not recur more than four times a week: microscopic examination of the urine affords convincing proof that there are no other emissions than those which occur during the night. Now, inasmuch as a young man may without any bad consequence accomplish coitus three or four times a week, it may be assumed that if another young man who has involuntary seminal emissions three or four times a week has also very serious nervous symptoms, the latter are not caused by the loss of semen.

Another fact has to be stated. Involuntary seminal emissions, are much less abundant than emissions preceded by the ordinary venereal excitement. On the other hand, microscopical examination shows, that in the seminal fluid of persons affected with spermatorrhœa, there are far fewer spermatozoa than in semen discharged *in coitu;* and physiologically, this ought to be the case, as the erotic excitement not only induces a more profuse secretion from the vesiculæ seminales, but likewise from the testicle itself.

Moreover, the intense nervous excitement which precedes, accompanies, and in a special degree terminates the act of copulation, leaves as its immediate consequence an amount of prostration which testifies to the great effect which it has produced on the nervous system: but nothing of a similar description is observed after involuntary seminal emissions, which frequently take place without erotic dreams, or after excitement so transient and little felt as sometimes not to be perceived by the patients except by the soiling of their bed or clothes.

Gentlemen, I do not know whether these considerations strike you as they strike me: but I am forcibly led to the conclusion that the nervous disorders observed in spermatorrhœal patients do not depend upon loss of seminal fluid.

By this statement, however, I do not mean to imply, that seminal emissions are without any influence on the system. I mean that in a hale young man, whose nervous system is in a state of harmonious action, coitus, terminating in seminal emission, may take place twice a week without any detriment to health, but if it be granted that in the spermatorrhœal patient, the brain and spinal marrow are in a state of debility, a smaller seminal loss, even when unaccompanied by sufficient erotic excitement, will become a powerful cause of nervous disorder; and this disorder will almost inevitably become excessive, if the seminal emissions recur very frequently, as is too often the case. Thus it is, that a bad state of the nervous system predisposes to spermatorrhœa, and that spermatorrhœa peculiarly aggravates the nervous affection which is the primary source of the evil.

Gentlemen, the symptoms which may bring in their train involuntary seminal emissions, by no means all show themselves in the same individual, neither do they develop themselves in the same order of succession, nor do they always reach the same extreme degree of severity. One or other phenomenon will in general pre-

dominate over the others, and so great will be its predominance, that the patient's attention will be directed to it exclusively. This may lead the physician, unless he be specially on his guard, to form an erroneous diagnosis, and to believe that there exists an affection very different from the real one. I cannot too frequently repeat, that the disease may present in its aspect, and in its progress, an infinity of varieties; and that its different forms are subordinate to individual peculiarities, and many special circumstances difficult to foresee. They are also subordinate to intercurrent complications.

In a young man, twenty-six years of age, whom we had in bed 18 of St. Agnes's ward, the disease was only of four months' duration, and seemed to have had a chronic gonorrhœa as its starting-point. The individual to whom I refer told us, that the gonorrhœa, of which he had only got rid nine or ten months previously, had lasted three years. The spermatorrhœa declared itself five or six months later, or at least only at the time when the patient first observed that he had involuntary seminal emissions on going to stool. Three weeks before the symptoms set in, he was troubled with obstinate constipation; but although the constipation ceased, and although the alvine evacuations became regular, and regained their natural consistence, the pollutions continued without abatement. From that time also, nocturnal pollutions supervened, an occurrence not in accordance with the general rule; for it is usual for the nocturnal to precede the diurnal pollutions. The nocturnal pollutions always occurred in connection with erotic dreams: the diurnal pollutions, which, as the patient stated, first took place when the bowels were being moved, and then recurred more and more frequently. If he attempted coitus, ejaculation occurred almost immediately, not only before copulation, but before there was complete erection: ultimately, even before there was any erection at all. This young man told me that one day when looking at the picture of an amorous couple exhibited in a shop-window, a pollution without previous erection was caused by the lascivious representation.

The general health of the patient was very much out of order. He complained of excessive debility; and was fatigued by the least exertion. Palpitation of the heart would be brought on by a rather long walk: his appetite became impaired, and he soon had a feeling of disgust for the different kinds of food which he tried to take: he preferred spiced alimentary substances and those prepared with oil or vinegar. Eating, however, temporarily calmed the gastric pain

which preceded a repast, and caused a cessation of the eructations, which were of sickly odour and accompanied the pain; but in two hours, both pain and eructations always returned.

Tonic treatment, sulphurous baths, cold hip-baths, and preparations of quinine (prescribed in one of the services of this hospital) produced some improvement. His disease, however, having soon become as bad as it was before, he returned to the Hôtel-Dieu.

The existence of gonorrhœa of old standing having suggested the idea that the spermatorrhœa might depend on a chronic affection of the urethra, an instrument was passed to ascertain whether there was stricture. The patient stated that he had experienced some difficulty in making water for the preceding three or four months—that the stream which was long in coming, was flat and spiral—that it was sometimes interrupted—and that some drops of urine escaped after he believed that he had completed micturition.

On introducing the sound, I encountered one obstacle at the entrance of the urethra, a second about the middle of its cavernous portion, and a third obstacle in its prostatic portion. The treatment indicated by Lallemand seemed suitable; but it was not tried, as the patient was dismissed, at his own request, after a residence of three days.

Gentlemen, I shall now recapitulate some of the conditions under the influence of which spermatorrhœa is produced—conditions with which it is essential to be acquainted, that we may be able to institute a rational treatment of the disease.

Here, I find it necessary to make a short digression into the domain of physiology.

To the question: *By what organ is the semen secreted?* it might be supposed that no other reply could be given than—"*By the testicle.*" Nevertheless, this is not the correct answer; as is proved by experiments on the lower animals, as well as by the facts observed in the human subject.

Take an animal—a young dog, for example—tie the deferent ducts in two places,—and cut them through between the ligatures. In this way, the testicles will be completely isolated from the urethra. Still, the animal will be able to copulate with energy, and the seminal ejaculation will be nearly as abundant as before the operation. A somewhat similar phenomenon is observed in the human subject. An individual contracts a gonorrhœa: this, to use

a vulgar expression, falls into the scrotum [*tombe dans les bourses*], or, to speak more in the language of science, there supervene inflammation and consecutive induration sufficient to cause obliteration of the excretory ducts of the epididymis; but this does not deprive the individual of venereal aptitude: he will be quite as able to copulate as before, and ejaculation will lose nothing in energy or power. This arises, Gentlemen, from the seminal fluid being furnished by two sources: the larger portion comes from the vesiculæ seminales and the other, and smaller portion, from the testicles. The portion, however, derived from the testicles is the most important, as it is that which contains the fecundating substance and the spermatozoa. In the pathological conditions of which I have been speaking, although the venereal aptitude remains, and although ejaculation can take place, the seminal fluid loses its essential properties, and if both epididymes are similarly affected, the individual is rendered unfruitful. These facts, of which Hunter caught a glimpse, have lately been wonderfully cleared up by Professor Gosselin.[1]

A knowledge of the physiological fact now stated will enable you to understand how it is that certain young men of sound and vigorous constitution, presenting, moreover, all the attributes of virility, are, nevertheless, unfruitful. This is a point regarding which it is specially important for the practitioner to be cognizant, as it is one on which he may be consulted. Before seeking for other causes of the sterility complained of, scrupulously interrogate your patient, attentively examine him, inquire whether he has not had at some former time an attack of gonorrhœa, and discover whether, as a consequence of that attack, he has not complete obliteration of the spermatic passages. This fact requires the more attention as at first it might appear that the condition of the individual was similar to that of a eunuch. His state, however, is very different: do not make any mistake on that point. The old man in the last stage of decay, who for many years has been incapable of sexual intercourse, impotent though he be, and consequently unfruitful, bears no resemblance to the man who has been castrated, for his useless testicles still give him the stamp of virility: so long as he retain them, he will retain certain attributes of manhood, such as the

[1] Gosselin:—Nouvelles Etudes sur l'Oblitération des Voies Spermatiques et sur la Stérilité Consécutive à l'Epididymite Bilatérale. [*Archives Générales de Médecine* for November, 1853.]

bass voice and the beard; but these attributes will at once disappear should he lose the testicles which have apparently ceased to perform any part in the economy. A very curious work of Professor Charles Robin upon sarcocele contains a demonstration of the correctness of this statement.[1]

A person has double sarcocele, and although it might be supposed that his testicles were destroyed, he does not really become a eunuch till after the surgeon has removed both organs. This is the light in which Professor Robin looks at such cases: they belong to the same category as the two cases of Professor Gosselin—in this sense, that in them there is no disease of the testicles. Sarcocele is an affection which pertains to the epididymis and remains separated by the *tunica albuginea* from the gland, the tissue of which it so much respects that the normal structure of the filamentary tubules is well preserved, although the tubules are generally found displayed upon the surface of the epididymotic tumour.

I have already told you, that the same thing has been observed in cryptorchidous persons: that is to say, that though they are unfruitful, they are not impotent.

That fact being well established, let us resume our subject. To enable a gland to perform its function, there is no necessity for the direct action on it of the stimulus. Irritation of the mucous membrane of the mouth, stomatitis, may induce abundant secretion from the salivary glands, though they are not implicated in the inflammation, just as inflammation of the ocular mucous membrane occasions in a similar manner a more copious flow of tears; just as irritation of the gastro-intestinal mucous membrane occasions a greater secretion of bile and pancreatic juice; or finally, just as irritation of the bladder, cystitis, is accompanied by a secretion of urine which is more abundant than usual, and which is also passed more frequently, even although, as in this case, the affected organ is far removed from the gland the functions of which it sympathetically excites. Well then, in similar sympathetic action, we find a cause of involuntary seminal losses.

It is upon this fact that Lallemand has based almost the whole of his theory of spermatorrhœa. He holds that spermatorrhœa is nearly always dependent upon irritation of the prostate gland and

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[1] Robin (Charles):—Mémoires de la Société de Biologie. Second Series. Vol. III, (for 1856): p. 1867. Paris: 1857.

its ejaculatory ducts: and he believes that in most cases this irritation, which also exists in the neck of the bladder, is the result of chronic inflammation of the urethra in the prostatic portion of the verumontanum. According to the illustrious professor of Montpellier, the most frequent cause of involuntary seminal emissions is an old attack of urethritis, or of gonorrhœa; and these seminal losses, he says, are often related to stricture of the urethra.

This class of causes does not admit of being called in question; and the sympathetic irritation which the chronic inflammation of the urethra determines may likewise have as a starting-point affections seated in organs communicating more or less directly with the vesiculæ seminales and ejaculatory ducts. In this way, affections of the rectum, among which may be mentioned hæmorrhoids, the presence of vermicular ascarides, and even—although much more rarely—lumbroid ascarides, may be the cause of spermatorrhœa.

I have stated that involuntary losses of semen are in some cases provoked by obstinate and habitual constipation; but in these cases, the seminal emission is induced mechanically by compression exerted upon the vesiculæ seminales by the excrementitious bolus, as it is being expelled with difficulty and great effort.

Though for the most part these are the causes which induce spermatorrhœa, undoubtedly there are also others. Thus, seminal losses may supervene under the influence of spasm, in a manner similar to that which we saw take place in nocturnal incontinence of urine. I shall afterwards have to address you in a more special manner upon this latter affection; but that I may enable you the better to accurately grasp my views on the subject before us, I shall now recapitulate what occurs in nocturnal incontinence of urine. The patients in whom this affection is observed—chiefly children—generally pass the urine during the day in a stronger stream than other persons: this greater vigour of emission in voluntary micturition shows a greater energy in the contractile power of the bladder. It is a still more curious circumstance, that children who cannot retain their urine in its natural reservoir during the night, can sometimes during waking hours retain it more easily, and for a longer period, than others. How are we to explain this fact, which was long ago pointed out by Bretonneau? During sleep, the bladder enters into a state of erethism, which may be compared to that of the external genital organs; for, as you are aware, there is always a

state of erection during sleep in children and young men. The cause of incontinence of urine is a condition analogous to that of erethism: it is due, if you will allow me to use such an expression—it is due to the vesical muscle itself entering into a state of erection. That this take place, it is not necessary that the bladder should be full, nor even that it should contain a great quantity of fluid: so far from that being the case, the urine is emitted as soon as a small quantity has accumulated in the bladder: and that it thus happens is proved by the fact, that it is during the early part of the night that the child is troubled with incontinence of urine. The child is made to pass his water when he is put to bed, and it is within two hours—not after a lapse of eight or ten hours—that he wets his bed. I repeat, therefore, that the occurrence is dependent upon a very energetic contraction, a sort of temporary erection of the vesical muscle, which the sphincter, charged with closing the orifice of the neck of the bladder, is unable to resist.

An analogous mechanical cause may produce involuntary seminal emissions. It is during the early hours of the night, that pollutions occur. Under some exciting influence, which at times is occasioned merely by the position in bed of the individual, the vesiculæ seminales enter into a state of erection; or, if you prefer the expression, contract in the same energetic manner in which I have said the vesical muscle contracts; and the result is an ejaculation of seminal fluid without the patient being conscious of the occurrence, just as in the other case, there is involuntary emission of urine.

Spermatorrhœa may be the result of atony of the organs which secrete the seminal fluid, as well as of their too energetic contraction. The state of the organs is then quite passive, just as it sometimes is in emission of urine when the incontinence occurs during the day. There are individuals, both children and adults, who are not able to retain their urine, when even a small quantity has accumulated in the bladder: in them, the jet is weak and dribbling, and not strong, as in the other class of persons of whom I have just been speaking. This incontinence depends on a debility of the sphincter, which is absolute, not relative, as in the class of cases previously described, in which the contractility of the vesical muscle is greater than that of the muscular fibres encircling the neck of the bladder. The same takes place in respect of involuntary seminal emissions: the ejaculatory ducts from atony, are unable to resist the feeble contraction of the vesiculæ seminales, and to retain the semen which is passing

through them, so that there is ejaculation, or, to speak more correctly, an involuntary flow of semen as soon as it is secreted. My comparison of this kind of spermatorrhœa with diurnal incontinence of urine is peculiarly appropriate, as it may be extended to the treatment: the same treatment is equally efficacious in both maladies, as you saw from a case of incontinence of urine which we had in St. Agnes's ward, a case of which I intend to speak when I come to address you regarding the latter malady.

Sufficient attention has not been directed to *impotence* consecutive to spermatorrhœa. How often have I been consulted by persons for their infirmity, and how many of these patients have frankly disclosed it; but how many more have only come to confess it after numerous circumlocutions! Well then, nearly all who were impotent from atrophy of the testicles, or from cryptorchidia, were found to have had nocturnal incontinence of urine in childhood, involuntary pollutions at puberty, and at the age of manhood, either inability to have an erection when with a woman, or if they had an erection, it was of so transient a character that the penis was hardly introduced ere the copulative act was completed by premature ejaculation, unaccompanied by any voluptuous sensation. Who does not perceive that the phenomena are of the same character, in the incontinence of urine of childhood, in the incontinence of semen of puberty, and in the genesic impotence of manhood? And who does not also perceive, that these successive infirmities are dependent on an imperfection in the nervous system of organic life? Some of these patients tell us that they are frantically addicted to masturbation: and they believe that their impotence is the consequence of these lamentable excesses. But is not the practice of masturbation an indirect proof of my proposition, that there is a bad state of the nervous system? Is it not in fact mental aberration which impels these poor infatuated creatures to their solitary debauchery? If, at a later period, these same persons become impotent, insane, or paraplegic, we must not conclude that this is the direct consequence of masturbation, but must rather regard it as an aggravation of a nervous condition of which masturbation was only the first morbid manifestation.

From the considerations into which I have now entered, there may be deduced certain therapeutic indications which must be borne in mind in treating spermatorrhœa.

Prior to the date at which Lallemand published his important researches, spermatorrhœa had enlisted the attention of physicians

only in a small degree—there was an incorrect understanding of its serious character, and of the disastrous consequences directly and indirectly induced by it: it was treated without method, and by purely empirical means.

Lallemand, by presenting the disease as almost exclusively the result of irritation of the spermatic ducts arising from chronic inflammation, showed that the treatment indicated consisted in modifying the condition of the mucous surfaces, the seat of that inflammation. He recommended, as the measure best calculated to attain this object, cauterisation of the mucous membrane of the prostatic portion of the urethra, so as to touch the verumontanum near the opening of the ejaculatory ducts. In support of his opinion, he adduces a great number of sufficiently apposite cases, so that when one reads his work, a conviction remains that the operation now mentioned is often useful. It is not necessary for me in this place to describe to you Lallemand's method of performing this description of cauterisation: in relation to that point, I cannot do better than refer you to his *Traité des Pertes Séminales Involontaires*, where you will find every possible information as to the most desirable way of carrying out the treatment.

However incontestible may be the utility of this medication in circumstances such as I have now described, Lallemand has in my opinion committed a mistake in generalising its application; and he has made that mistake, because he would also generalise beyond measure the influence of urethral inflammation upon the production of involuntary seminal emissions. Let me repeat then, that while the utility of cauterisation is incontestible in the circumstances which I have pointed out, that is to say in spermatorrhœa arising from inflammation of the urethra, it is not applicable in other kinds of spermatorrhœa, in which an entirely different kind of treatment is required. In them, it is the spasmodic element against which we have to contend; and belladonna, so marvellously useful in nocturnal incontinence of urine, is employed to render real service in that form of spermatorrhœa which presents a certain analogy to nocturnal incontinence of urine. Belladonna, however, is far from being as efficacious in the former as in the latter affection: still, the cases of spermatorrhœa in which this medicine has seemed to me particularly useful are so numerous that I recommend you to give it a trial.

The utility of belladonna in spermatorrhœa, as well as in nocturnal incontinence of urine, does not perhaps depend upon that

obtunding influence which it exerts upon the contractility of the vesiculæ seminales, but upon its undoubted influence upon the entire nervous system, particularly on the encephalon and spinal marrow. Its great power is shown by the undoubted special effects obtained from it in epilepsy, tetanus, and many other nervous diseases.

Probably, to a similar special influence is due the real service obtained by preparations of digitalis and aconite in the treatment of spermatorrhœa. To a similar kind of property are attributable the favourable results of my using internally the nitrate of silver, a medicine which certainly produces no stupifying effects.

Believing that there is always spinal irritation, or at least an altered state of the spinal marrow, depending perhaps on congestion of the cord, I frequently advise repeated recourse to dry, or even sometimes to sanguineous cupping over the spine, to the application to the same situation of lotions of tincture of iodine, and a thick woollen stuff impregnated with embrocations of essence of turpentine, over which a very hot iron is passed. Finally, I do not hesitate, in certain cases, to apply moxas and flying cauterics: I act thus energetically particularly when pains supervene in the limbs, the first symptoms of locomotor ataxy, a terrible disease, the existence of which is often announced by spermatorrhœa.

Hydropathy, as an antispasmodic agent, ought to occupy an important place in the treatment of seminal losses; and sea-bathing, really only another form of hydropathy, is likewise exceedingly beneficial.

When spermatorrhœa depends upon an excess of energy in the contractions of the vesiculæ seminales and ejaculatory ducts, I prescribe *warm hip baths,* directing them to be used as hot as the patient can take them. I also recommend *bags of hot sand* to be applied to the whole of the perineal region: the application ought to be made at night when the patient goes to bed, and it ought to continue each time at least half an hour.

It will no doubt surprise some of you to hear me recommending hot baths, when most physicians order cold baths in the treatment of spermatorrhœa. Perhaps I may be accused of acting in a spirit of contradiction, while in truth I have no such tendency, being always inclined to accept, come from whomsoever it may, the oddest possible modes of treatment, provided their application appears to be devoid of all risk. While, therefore, I now speak in favour of warm baths, while in a more general way, I state my conviction that hot

are preferable to cold applications in the class of cases now before us, I have my reasons for so doing. On many occasions, I have pointed out to you the great power of caloric as an antiphlogistic agent, and in contrast, have mentioned the potency of cold as an energetic excitant. The truth of these propositions is absolutely demonstrated by the most commonly known facts. When the hands are immersed in snow, or in iced water, the chill felt in the first instance is soon succeeded by a great increase of temperature: when the hands are immersed in very hot water, in water sufficiently hot to cause temporary congestion of the tissues, the congestion is quickly followed by a fall of temperature, and a notable diminution in the colour of the skin. It happens thus, because the ultimate effect of the application of heat is sedative, while that of cold is eminently phlogistic. The sedative power of heat is frequently made use of in medical practice. This is an important question, the complete discussion of which I reserve for a future day. In the mean time, you will understand why I recommend the application of bags of hot sand, and hip baths, as hot as the patient can bear them, in the treatment of spermatorrhœa. I must inform you, however, that, in the first instance, there is an increase of the symptoms which we are seeking to subduc; but this temporary increase of excitement is of short duration, and amendment is not slow in showing itself.

Although hot hip-baths are useful in those cases of spermatorrhœa in which there is augmented excitability and contractility of the vesiculæ seminales and ejaculatory ducts, they are injurious in that form of the malady which I have designated *passive*. In passive spermatorrhœa, cold bathing, that is to say *hydropathy* is clearly indicated. It is unnecessary to recapitulate what I have just been saying to explain to you the manner in which the treatment by the employment of cold acts beneficially in the passive form of spermatorrhœa.

In addition to the use of cold, there are certain medicines which ought to be administered internally in this class of cases. Among them, the foremost place is taken by nux vomica, St. Ignatius's bean, and the preparations of strychnia. These medicines, begun in minute doses, ought to be steadily and gradually increased, till they produce their physiological effects.

Nevertheless, Gentlemen, it sometimes happens that all these means fail. Another, however, remains to be tried, one which I have been in the habit of employing for a great many years, and

sometimes with success. I refer to compression of the prostate gland. Let me now describe the manner in which I employ this mechanical means of treatment.

In 1825, when I was attached as *interne* to the *maison de santé* at Charenton, Dr. Bleynie, the physician attached to the institution, mentioned to me the case of one of his patients, who had suffered from impotence, and had been cured by wearing, by the advice of a charlatan living in the Place Baudoyer, Paris, a sort of box-wood pessary in the rectum. The fact seemed to me strange; and at that period, like everybody else, not understanding the relation of impotence to spermatorrhœa, I was quite unable to comprehend how a cure could be accomplished by means so singular. I only saw in the proceeding a lascivious manœuvre similar to those practised in bad resorts by libertines seeking to restore artificially for the moment their exhausted virile powers. I did not trouble myself to seek for any other explanation; and the fact mentioned by Dr. Bleynie did not appear to me of much importance. Ten years later, however, I myself used the same empirical treatment; and in then reflecting upon it, I formed a theory as to its mode of acting. I was treating a young man, aged 26, who was suffering from involuntary seminal emissions and absolute impotence. This unfortunate individual was on the point of marrying, and you can understand how this circumstance increased the state of melancholy into which he had already been thrown by his malady: suicidal ideas were passing through his mind. His malady had resisted all the means by which I had endeavoured to cure it, when, recollecting the case mentioned to me by Dr. Bleynie, I resolved to try it as a forlorn hope. I advised the patient—who spent the whole day sitting on a chair in his room—to wear within the anus an apparatus which I caused to be made for him. It was a sort of box-wood stem-plug resembling that of a speculum: when introduced into the rectum, it was kept in position by means of towels. After fifteen days of this treatment, the young man came to see me, when he informed me to my great astonishment, I confess), that the means had been, to a certain extent, successful. The virile aptitude began to reappear as the frequency of the seminal emissions diminished. Encouraged by this result, I recommended the patient to persevere in the use of the same means. In a fortnight, the cure was sufficiently complete to allow the patient to marry. Being entirely in his confidence, he told me, that he was able as any man to perform the conjugal act.

This first case gave me much to reflect upon, and set me, as I have just been saying, to seek for an interpretation of the cure: I asked myself;—how can impotence be cured by such strange treatment? It occurred to me that the stem pessary in the rectum acted beneficially by compressing directly the prostate, and indirectly the ejaculatory ducts; and that this pressure compensated for the deficiency of normal contractile power in the vesiculæ seminales. Having formed this theory, I set myself to verify it when similar cases presented themselves. Such cases have frequently occurred in my practice; and although this singular treatment has not always given me the looked for result, I can at least say that in a very considerable number of cases, the success has equalled my expectations.

Here, Gentlemen, is the apparatus which I am at present in the custom of using!

In principle, I have exactly adopted the bandage which persons with large hemorrhoids employ to prevent the profuse bleeding from which they so often suffer. The apparatus consists of a sort of small cone made of ivory or vulcanised caoutchouc fixed by a T bandage, which bandage is applied round the loins and kept in its place by a cincture to which are attached in front, bands passing under the thighs. The stem-pessary of my apparatus is longer and more bulky than that of the bandage used for compressing hemorrhoids, as it is requisite to be introduced higher up the rectum, so as to reach the situation of the vesiculæ seminales. I now use a simpler apparatus—simpler to this extent, that it does not require to be kept in its place by a bandage—an apparatus invented by Mathieu, our maker of surgical instruments. It consists, as you see, of a sort of metallic bung, of the form of a very elongated olive, varying in size, between a pigeon's egg and a small hen's egg. This bung diminishes downwards, taking the form of a neck, the diameter of which does not exceed five millimeters [about three sixteenths of an inch], so that when once introduced into the rectum, it is retained there by the natural constriction of the sphincter of the anus. The bung-like compressor is soldered upon a flat stem of the same metal about three or four centimeters in length and half a centimeter in breadth: the anterior half of the stem is intended to be applied to the perineum, and the other half to the coccygeal region. You see, Gentlemen, that this apparatus is of marvellous simplicity, and that when it is once put in its place, it cannot become spontaneously

displaced: moreover, when once it has been introduced, patients can wear it with ease continuously for a night and a day, without any necessity to fix it by a bandage. Of course, the size of the instrument must necessarily vary with individual peculiarities, with age, and with the manner in which its presence is tolerated.

I have told you, Gentlemen, that that part of the compressor of the prostate which is introduced into the rectum is soldered to a stem intended to protrude externally. This junction is effected in such a way as to prevent the two parts of the apparatus from being perpendicular, and so as to form by their union an acute angle of 75 degrees on the one side, and on the opposite side an obtuse angle of 125°. It is absolutely necessary, in placing the instrument, that the obtuse angle look towards the coccyx, and consequently, that the acute angle look towards the pubes: in this way, the superior part of the bulge will of necessity rest on the prostate.

I ought to add, that the length of the internal part of the instrument must necessarily somewhat vary. Digital examination of the prostate, in even a few cases, will suffice to make you acquainted with the fact, that it is situated at a depth varying between two and five centimeters, according to the height and greater or less degree of corpulence of the individual.

I cannot too often repeat that this apparatus has rendered me essential services. I have more than once seen involuntary seminal emissions, which had proved rebellious under every kind of treatment, yield completely, within a few days, under the use of this apparatus: after seven or eight days of its employment, the amendment has been such, that there was not only a rewaking of virile aptitude, but likewise a notable diminution in the general symptoms and mental disorder which accompanied the impotence.

On a future occasion, I shall tell you, that in certain cases of incontinence of urine, I have also found this treatment indicated. In the mean time, let me add, that it is equally useful in both the forms of spermatorrhœa of which I have spoken.

I have now described the means which I employ in the treatment of spermatorrhœa. Quite understand, that I do not pretend that these means are infallible; and still less suppose, that I pretend to cure the serious disorders of the nervous system which too often follow in the train of the malady! Even when the symptoms which have been its starting-point have entirely subsided, the supervened

perturbation of the functions of innervation, and the mental disorder will continue, and will resist all the efforts of art. On the other hand, the cases in which there is a definite cure, or at least a greatly ameliorated state consequent upon treatment, well planned and regularly carried out, occur, thank God! sufficiently often to be sources of encouragement.

I insist upon the necessity of a plan of treatment being *regularly* carried out; for when spermatorrhœal patients too quickly abandon the different means of treatment which I have mentioned, on account of the benefit obtained leading to a belief that the malady is radically cured, the amendment is not maintained, and ere long the symptoms reappear. It is necessary, therefore, that the treatment be persevered in: and this is all the more easy, that it is neither painful nor difficult to carry out.

It must not be forgotten, that an individual who has once been affected with spermatorrhœa is, from that circumstance, more liable to it than another who never before suffered from it. It is necessary, therefore, to take every possible precaution to prevent relapses. For this reason, the remedies to which the symptoms yielded ought to be persevered in for some time after recovery. The patient ought to be advised to revert to the compressor twice or thrice a year, for a fortnight or a month each time: he ought also, according to the indications, from time to time to recur to the hip bath, cold or hot, and to hydropathy. The medication I recommend will not, I repeat, prove efficacious, unless it be carried out for a long period, and in a methodical manner. Even when it is employed with extreme patience, failures will too often occur.

A few words more ere I conclude!

In September, 1863, a young Irishman came to me, recommended by one of my honorable professional brethren of Dublin. For two years, he had had involuntary seminal emissions during the night. In consequence of my friend Dr. Adolphe Richard having mentioned to me the good results he had obtained in similar cases by *forcible dilatation of the anus*, I entrusted the patient to him. He operated in my presence. From the day on which the operation was performed, there was no recurrence of the emissions; and four months later, in January 1864, Dr. Richard received a letter from the young man, in which he intimated that the cure still continued perfect.

In what manner does dilatation of the anus act in such cases?

This is a question which I cannot answer; but as the operation is totally exempt from danger, it ought to be added to the list of means which one may employ in the treatment of an affection so often rebellious against treatment as that which has formed the subject of this lecture.

LECTURE LXIII.

NOCTURNAL INCONTINENCE OF URINE.

Different Kinds.—Nocturnal Incontinence of Urine not a Morbid State in Lazy and Timid Children.—Nocturnal Incontinence of Urine (properly so called) is an Affection of the Nervous System, specially manifesting itself in an Excess of Excitability and Tonicity in the Muscular Coat of the Bladder.—Nocturnal and Diurnal Incontinence Coexisting depend on Atony of the Sphincter of the Bladder.—Treatment: Belladonna in Nocturnal Incontinence: Strychnine in Coexisting Nocturnal and Diurnal Incontinence.—The Prostatic Compressor.

Gentlemen:—You have frequently heard me interrogate a young girl who assists the nurses in St. Bernard's ward, as to the length of time she had been in the habit of wetting her bed. This girl, to whom I have more than once called your attention, was admitted to the clinical wards, about eighteen months ago, for nocturnal incontinence of urine. Under the influence of treatment by belladonna, the symptoms have gradually ameliorated to such a degree that the cure may now be looked upon as certain.

This patient, now 19 years of age, though presenting every appearance of a vigorous constitution, is of an eminently lymphatic temperament. During her residence in the hospital, we have several times had to treat her for attacks of scrofulous ophthalmia, leaving slight specks on the cornea, which have now, however, nearly disappeared. With the exception of these ophthalmic affections, and the malady for which she came into the hospital, her health has generally been good. Some months ago, she contracted pharyngeal diphtheria, when attending upon the diphtheritic patients in the nursery ward; but this diphtheria, although it occasioned some anxiety, was not followed by any bad consequences, and afterwards she soon resumed her ordinary good health.

According to the statement of this young girl, her incontinence of urine began when she was eight years of age. Till then, from early infancy, she had had nothing of the kind; and (repeating a statement of her parents), she said that her nocturnal incontinence of urine dated from a great fright which she had experienced. We shall see how much is attributable to this influence. After the fright, no night passed during which she did not wet her bed at least once, and occasionally two, three, or four times. It is a remarkable circumstance of which it is important to make special mention, that during the day and when awake, she could retain her urine as well as any one, and that it was only during sleep that she was unable to do so. Another point worthy of note is, that the irresistible necessity to pass urine seized her in the early morning during the last hours of sleep. This fact can be explained in a few words. The patient informed me that her sleep was deepest towards morning. It was then so profound that it was exceedingly difficult to awake her. When loudly called and well shaken, she neither heard nor felt anything; and when compelled to get out of bed, she seemed still to be asleep when on her legs. During the first hours of sleep, she often got up to satisfy a desire to pass urine; but whether she rose or not, she urinated during the night—and several times in the night, the nurse wakened her to prevent accidents—she did not the less wet her bed before morning, even when two hours had scarcely elapsed since she had last voluntarily relieved her bladder.

These details are interesting; and I shall return to them. But let us conclude the history of our patient. Immediately upon her coming into the hospital, she began to take belladonna. The effects of the medicine were at once manifested in a decided manner; and from the very commencement of its use, the frequency of the wetting the bed was reduced to once a night: subsequently, by increasing the doses, she passed several nights without wetting her bed: and at the present time, more than two months have elapsed since such an occurrence took place.

At the same time that this girl was under treatment, we had, in bed 3 of the same ward, a girl, 16 years of age, who was also affected with incontinence of urine. Her infirmity dated from birth. She told us that she generally went to bed at half-past seven in the evening, and wet her bed between eleven o'clock and midnight. She stated that when she slept during the day, no such accident occurred, because she then awoke from feeling the sensation of the desire to

make water. She added, that this desire whenever felt was always so urgent, that whether sleeping or awake, she had hardly time to run to the closet. I had recourse to belladonna, and with results as successful as in the other case.

In some cases, however, belladonna has completely failed me; and it has sometimes proved equally unsuccessful when combined with opium, or strychnine. Preparations of strychnine, however, are indicated in certain cases. I have not been more fortunate in the case of a young man in St. Agnes' ward with the recently lauded remedy, mastich. In him, however, the incontinence of urine ceased entirely after a small surgical operation.

This young man, aged 17, had, from childhood, been in the habit of wetting his bed: and the occurrence took place two or three times every night. No modification of his infirmity took place at puberty: but then, although his procreative powers seemed very slightly developed, the incontinence of urine became complicated with nocturnal pollutions. For a short time, I believed that the belladonna would have produced the benefit expected from it: in fact, the incontinence of urine seemed to have yielded; but this took place concurrently with profuse diarrhœa occasioned by the medicine, and it was easy to see that if the patient did not wet his bed, it was because he had to get up to stool frequently during the night. After unsuccessfully trying the syrup of the sulphate of strychnine, and mastich, it occurred to me that his congenital phymosis might be the cause of his infirmity. I therefore asked my colleague, Professor Jobert, to perform the operation of circumcision. From the date of the operation, thirteen nights passed without his wetting his bed, then for three consecutive nights he did wet it: finally, there was no return of the nocturnal incontinence of urine during the last nine nights he spent in the hospital, so that when he asked to be allowed to return home, permission was granted under the hope that he was definitively cured.

In bed 1 of the same ward, you lately saw a man in the prime of life, who was affected with incontinence of urine, and in whom the incontinence existed by day as well as by night. This individual was 51 years of age, and by trade a house-painter. He stated that he had had five attacks of painter's colic, but that since he was two years of age, he had never had any paralytic affection.

Two years ago, he felt that there was a diminution of strength in his legs, but this was not accompanied by any decrease of cutaneous

sensibility. This state of semi-paralysis, however, was general. The arms began to be affected: the tongue began to lose its freedom of motion, which produced a certain amount of difficulty in speech: the sight became enfeebled. But the symptom which gave this man most uneasiness, and was the principal cause of his coming into hospital, was the incontinence of his urine. Four or five times during the day, within a space of twelve hours, he was obliged to leave his work that he might make water; and at these times, he had no power to retain it. During the night, he wet his bed several times, because he felt no sensation, as when awake, of the necessity of emptying his bladder. Ultimately, this patient died in our wards of saturnine disease of the encephalon.

The form of incontinence of urine with which this man was affected has no relation to that other form of which I have been speaking. I was nevertheless unwilling to omit reference to this case, as I wished to call your attention to the therapeutic means which were employed.

Gentlemen, if nocturnal incontinence of urine cannot be regarded as a serious disease, it constitutes, at all events, a distressing infirmity, which you will frequently meet with in practice, and regarding which, moreover, any of you may be consulted at the very commencement of your medical career. I must not, therefore, allow to pass without notice the facts which are presented to your observation; and I must specially insist upon that mode of treatment which was so wonderfully efficacious in the first of our patients, and which has likewise generally proved successful when I have employed it. But before I give you the rules for carrying out this treatment, let me specify the circumstances in which it is indicated, or in other words, state the conditions under which nocturnal incontinence of urine is produced.

There are persons, particularly children, who piss their beds, dreaming that they are pissing against a wall or into their chamber-pot, the dream being suggested by a tormenting desire to make water, which desire they satisfy without awaking. There are others —children—who piss their beds from laziness: not wishing to get up when they experience the first intimation of the want to make water, they fall asleep again, and soon lose the power of retention. To this category belong cowardly folk, who, afraid of the darkness of night, neither care to leave their beds, nor call those who might come to their assistance, preferring to wet their beds to giving

themselves trouble. In this description of nocturnal incontinence of urine, an affection of much less frequent occurrence than is generally supposed, the accidents recur at long intervals. To cure such cases, there is no necessity for medical intervention: it is generally sufficient to exercise a moral influence over the patients—when they are young children, to threaten them with chastisement, and when they are older, to make them feel ashamed of themselves. It is altogether otherwise in respect of the nocturnal incontinence of urine now before us; and which is the result of a condition unquestionably morbid.

This affection, though it sometimes dates from birth, as in the young girl occupying bed 3 of St. Bernard's ward, does not generally supervene till the patient has attained a certain age. Like nearly all children, she wet her bed till she was fifteen or eighteen months old; and also, like most children, on attaining that age, she lost the habit; but then, after a time, it all at once returned. Generally, it is between the ages of seven and eight, that nocturnal incontinence of urine declares itself. In these cases the occurrence takes place nearly every night, and occasionally, several times during the same night.

What are the causes of nocturnal incontinence of urine? It is not unusual both for patients and their relations to state that the infirmity originated in a fright. You cannot be too much on your guard against accepting this explanation. Moral emotions, particularly fear, are too readily put forward to explain the origin of certain neuroses, although in reality, there is nothing to show that a relationship exists between them and their pretended causes. If a child has had epileptic fits, the parents will probably tell us that the first seizure was consequent upon a great fright. I do not deny that this statement may be quite correct; and in my lectures on epilepsy I have carefully pointed out to you the share which mental emotions have in the production of that dreadful malady. The share is relatively very small, and in one hundred cases in which this morbid cause is adduced, there may perhaps not be one in which it has had the least effect. On examining into the hereditary antecedents of the patient, you will find reasons more than sufficient to explain the existence of the malady.

It is well known that in respect of incontinence of urine, as in respect of many other neuroses, heredicity plays its part in a way which is indisputable.

Some time ago, I received in my consulting-room a young lady of 20 who had this infirmity. Almost every night wetting of the bed occurred during the first hour of sleep; but from that time till she awoke, there was no water passed. During the day, she had the power of retaining the urine for a very long time. Her mother, by whom she was brought to me, told me that she herself had suffered from incontinence of urine up to the age of puberty, when it spontaneously ceased; and that her son, up to the age of twelve, had had the same infirmity.

In July 1860, a lady, aged 40, brought me her son, who was about to go up for his examination for admission to the military school of St. Cyr: she stated that he had had nocturnal incontinence of urine since he was seven or eight years old. The symptoms, however, did not show themselves more than once or twice a month. This, however, was sufficient to render residence at a military school impossible. The lady informed me that she herself had had the same infirmity till she was twelve years old: she was then a very tall girl, and had attained puberty a year previously. Her mother, who had reckoned on puberty bringing with it a cessation of the incontinence of urine, finding that it was not so, became persuaded that her daughter did not sufficiently set herself to overcome the infirmity, and, acting on this belief, inflicted one morning in presence of the ladies' maid one of those chastisements which are hardly ever administered except to very young children. This punishment produced a deep impression on the girl's mind; and from the date of it, she never again wet her bed.

Similar cases are recorded. It is here, Gentlemen, that incontinence of urine and epilepsy present a certain point of contact. It is not at all unusual to meet with cases of epilepsy in the hereditary antecedents of the patients: it is not an overstrained inference to see in the incontinence of urine one of those transmutations of one neurosis into another, to which, upon several occasions, I have called your attention. My view is rendered the more tenable by the fact, that the transmutation sometimes occurs in the same individual, as in some cases which I have brought under your notice. In one of the cases to which I allude, the patient was a child who had been in the habit of wetting his bed till he was nine years old; and who, when cured of that infirmity, became subject to epileptic fits. Hysteria very often plays the same part as epilepsy in these transmutations. Finally, when lecturing on spermatorrhœa, I pointed out the

relationship which exists between that affection, nocturnal incontinence of urine, and the neuroses: I showed you that in a great many cases, seminal incontinence succeeded at puberty to the urinary incontinence of childhood, and that impotence at the age of manhood was the accompaniment of seminal incompetence. Then, again, to crown the whole, you have seen epilepsy or insanity terminate the morbid series, thus proving, that each of its parts is of the nature of a neurosis.

I have said, that we must admit our ignorance of the causes—I mean the exciting causes—of incontinence of urine. In some cases, as in that of our young lad in St. Agnes's ward, phymosis was the starting point of the morbid symptoms: this fact may be explained by the sebaceous secretion between the prepuce and the *glans penis*, causing an irritation extending by sympathy to the bladder, and rendering that organ more easily excited to contraction by the accumulation of urine in its cavity. Cases of this kind, however, are exceptional; and as a general rule, it may be stated, that the immediate cause of the infirmity cannot be detected.

Perhaps we are better acquainted with the proximate or organic cause of incontinence of urine.

The opinion was long ago stated—and it is still held by some physicians—that this infirmity results from feebleness of constitution: that the subjects of it are weak children of lymphatic temperament, flabby flesh, pale visage, and fair hair. A more attentive examination of the facts will show you, that this opinion is too exclusive. No doubt, nocturnal incontinence of urine is met with in subjects of delicate constitution who are devoid of moral and physical energy; but then, it is met with nearly as frequently in persons possessed of every attribute of vigour, and in the enjoyment of perfect health.

Not to pursue this topic any farther, I repeat, that nocturnal incontinence of urine is a neurosis; and I now add, that it is a neurosis manifesting itself by excessive irritability of the bladder. In fact, the immediate cause of incontinence is this excess of irritability in the muscular fibres of the bladder. Such is the conclusion I have arrived at, in seeking to explain to you the success obtained in treating this affection by belladonna: it is one proof more of the truth embodied in the aphorism of Hippocrates:—"*Naturam morborum curationes ostendunt.*"

You know, Gentlemen, what takes place in the act of micturition.

The urine secreted by the kidneys flows by the ureters into the bladder, where it accumulates, and is there retained by the obstacle to its egress presented by the sphincter. When a greater or less quantity of urine has been accumulated in this manner, the sensation of a want to urinate is felt, which sensation is due to the muscular fibres of the bladder contending against the resistance offered by the sphincter. Although this sphincter, like all the muscles of organic life, is placed external to the domain of the will, its contractions may nevertheless be rendered more energetic by an exercise of the will, so as to enable the individual at pleasure to retain the urine for a longer or shorter period: the constriction of the neck of the bladder by the fibres of its own sphincter is increased by the contraction of the *acceleratores urinæ* and the *levator ani* muscles. But at last, the want to urinate becoming more pressing, in consequence of the greater accumulation of urine, the retaining, are overcome by the expulsive forces, and micturition takes place. It is the result simply of the contraction of the muscular fibres of the bladder: this action is generally sufficient to empty the bladder, particularly when the sphincter offers little resistance, as is the case in very young children. In older children, and in adults, this action is not always adequate, and then the diaphragm and abdominal muscles assist in micturition.

It is necessary, therefore, for the accomplishment of voluntary micturition, that the resistance of the vesical sphincter be sufficiently powerful to counterbalance the action of the muscles which tend to expel the urine from its natural reservoir.

Should the resistance of the sphincter not be sufficiently powerful, the urine will escape involuntarily—there will be incontinence.

This defective resistance may be either absolute or relative. It may be absolute, as in cases of paraplegia and paralysis. There was an absolute deficiency of resistance in the patient who occupied bed 1 of St. Agnes's ward, who, as you will recollect, suffered from saturnine symptoms, and had paralysis of the bladder. But under such circumstances, there will not only be symptoms of nocturnal incontinence, but also an involuntary escape of urine by day as well as by night; and as the bladder itself will participate in this paralysis, the flow will take place in a passive manner, and not in a strong stream, as observed in the case at present specially under consideration.

In nocturnal incontinence of urine, the deficiency in the resisting

power is only relative, inasmuch as then it is the irritability of the muscular fibres of the bladder itself which is augmented.

This irritability, and let me add, this exaggerated tonicity of the bladder, are demonstrated by the fact stated by Bretonneau, a fact which I have also verified, that the majority of the patients suffering from nocturnal incontinence of urine piss by day in a very strong stream. This irritability appears to me to be also demonstrated by the fact, that when the patients are asleep, the penis is almost always is a state of erection. May it not be assumed, therefore, that the bladder participates with the external genital organs in a condition of erethism ?

Along with the augmented tonicity of the muscular fibres of the bladder, there may exist a certain degree of atony of the sphincter, and then the patients, as in the case of the young girl of bed 3, St. Bernard's ward, have great difficulty in retaining their urine even during the day. But this, let me say at once, is an unusual occurrence : the vesical sphincter, at least when under the control of the will, preserves its power intact. What shows that here we have nothing to do with atony of the vesical sphincter is, that the individuals when awake are able to retain their urine for a very long time. The young girl, the subject of our first case, told us frequently, that it was so with her. Now, is it possible to explain the existence of this power, if we admit that the contractility of the sphincter is enfeebled ?

The incontinence is nocturnal : the incontinence supervenes during sleep, and when sleep is most profound. Interrogate the patients, or rather interrogate the persons who can give information regarding them, and you will learn, that persons who wet their beds are generally deep sleepers. Such being the case, it is easy to understand the phenomena.

During sleep, the voluntary contractility of the vesical sphincter being completely annihilated, its organic contractility is insufficient to contend against that of the muscular fibres of the body of the bladder, and consequently involuntary micturition occurs. To produce this effect, it is not necessary that there should be any great accumulation of urine.

Let us now return to our patient in St. Bernard's ward. Although she got up several times during the night to make water, either awaking spontaneously, or being awoke for that purpose so that she might avoid wetting her bed, this occurrence was not prevented, the

wetting the bed still taking place towards morning during her last hours of sleep. This is what happens with many of those affected with this infirmity. There is, therefore, no ground for supposing, as do certain authors, that nocturnal incontinence of urine is due to excessive distension of the bladder, and that the overflow is the result of engorgement, as it is generally in retention.

Gentlemen, connected with the question now before us, there is an important point which requires to be elucidated.

The young girl in St. Bernard's ward sensibly improved from the very commencement of the treatment which I prescribed. After taking the belladonna for a fortnight, she was ten or twelve nights without wetting her bed, whereas previously, she had done so every night at least once, and often twice. Meanwhile, her catamenia having appeared for the first time, the nursing-sister, believing that a crisis had come which would radically cure the infirmity, discontinued the administration of the medicine. The incontinence soon returned.

The opinion is pretty prevalent even with physicians as well as with the public, that the establishment of the menstrual function, and, in a more general way, the influence of puberty, are curative of nocturnal incontinence of urine: there is also a prevalent notion, that when the infirmity dates from birth, it will yield naturally at the period of the first dentition, or if not then, at the second; and in the same way, if the period of puberty pass without a cure, it is hoped that marriage, or at all events the birth of the first child, will lead to the desired result. Gentlemen, beware of such illusions! If you participate in them with the family of the patient, when they seek your advice under the circumstances, you will expose yourselves to very awkward disappointment. Unquestionably, as incontinence of urine often ceases spontaneously, so may this cessation take place at some particular epoch, such as that of dentition, puberty, marriage, or the first childbearing: but I regard such an occurrence as a mere coincidence, judging from numerous cases which I see every day, in which the symptoms remain unchanged, although the various changes referred to take place in the organism.

Once, however, I think I met with a case in which there was an evident relation between a first pregnancy and the cure of nocturnal incontinence of urine. The patient was a girl of 18, who, from birth, had been in the habit of wetting her bed. A vain hope had been entertained, that dentition would lead to recovery: at 14, men-

struation was established, but there was no amelioration: some one then told the family that marriage would accomplish a cure. The young lady possessed all that was desirable in respect of beauty and fortune: but how could she be given in marriage without telling the man of her infirmity? And where was to be found a conscientious man who would incur the hazards involved in such a marriage? There was found, however, an individual without fortune, who did not shrink from the match. He married the lady, who immediately became pregnant; and ceased from that time to wet her bed. This is the only case of the kind with which I am acquainted: and therefore, I cannot say too decidedly, that such cases are altogether exceptional.

Intercurrent diseases occurring in the subjects of incontinence of urine have an influence upon that infirmity. Acute and febrile diseases, particularly the eruptive fevers, whilst they continue, suspend the manifestations of incontinence; and this beneficial influence is sometimes prolonged for some time after the fever has ceased. You, of course, recollect the young girl who was the occupant of bed 22, St. Bernard's ward. I treated her fruitlessly for eighteen months. In the summer of 1861, she had an attack of dothinenteria, and during her convalescence, she passed twenty-four nights without wetting her bed. At a later date, the habit returned. Do you not perceive in these facts an additional feature of analogy between nocturnal incontinence of urine and the other neuroses?

Gentlemen, many measures have been tried for the cure of incontinence of urine, which although it be not a serious affection, and though it be one which generally ceases spontaneously, is nevertheless a deplorable infirmity which medical practitioners are constantly being required to treat. Among the therapeutic agents which have been employed, belladonna (or atropine) occupies the first place. When administered under the conditions existing in the majority of cases, and in accordance with certain rules, it constitutes an eminently useful, nay almost an infallible remedy. It almost always notably diminishes the frequency of the bed-wetting, and in a large proportion of cases, causes it entirely to cease.

The treatment by belladonna, which I adopted from the practice of Bretonneau, has for a long period rendered me undoubted service; and similar benefits have been obtained from it by Dr. Blache and others. You yourselves have been witnesses of its successful employment in the case of the first of the young girls in St. Ber-

nard's ward. I shall give you a summary of that case, and recapitulate the rules of treatment which I followed.

On her coming into the hospital, I caused this girl to take every night a centigramme [one seventh of a grain] of the extract of belladonna, in the form of pill: obvious improvement was the immediate result. In place of wetting her bed twice every night, she did so only once: and whilst she went on taking the medicine, without increasing the dose, the occurrence no longer took place every night as before. She passed three or four consecutive nights without wetting her bed: she then resumed the habit, wetting it perhaps from two to six nights in succession. That state of matters continued for several weeks. Not allowing myself to be discouraged by the obstinacy of the affection, I increased the doses of the belladonna, running up the quantity by degrees to ten centigrammes [one grain and three sevenths]. The dose was taken only once in the twenty-four hours: and night was always the time at which it was administered. Under the influence of the dose of ten centigrammes, the amendment was so great, that the patient passed twenty-two consecutive nights without wetting her bed. She had then a relapse for two nights, after which, eight or ten nights elapsed without any recurrence: she then wet her bed for two or three nights in succession, when again, for ten nights, the accident did not recur.

We had assuredly made way; but were still far from having attained a cure. I persevered in the treatment, and increased the dose of the extract of belladonna to fifteen centigrammes [two grains and one seventh]. During the last fifty days, the patient has been taking the nightly dose of fifteen centigrammes; and during the whole of that period, she has not once wet the bed.[1]

Gentlemen, this case shows you that when experience has convinced a physician of the utility of a mode of treatment, he ought to persevere in its employment. In respect of the matter now before us, let me repeat my profound conviction that *belladonna* is the most powerful therapeutic agent in cases of nocturnal incontinence of urine in both sexes. Though I am not absolutely certain that I

[1] Subsequent to the delivery of this lecture in the theatre of the Hôtel-Dieu, no relapse occurred: that the cure was really radical can be affirmed with certainty, as the patient remained in the hospital till the end of 1863, as an attendant in the wards.

shall invariably cure my patients by this heroic medication, I feel quite sure that I shall almost always afford them a great measure of relief. Strong in my conviction of obtaining a successful result, I carry out the treatment of the case with patience, and I claim that same patience from those confided to my care: in the case which you have seen me treat, it is for you to say, whether the result obtained corresponded satisfactorily to the attempt. Unfortunately, it is not always possible to get the patients to continue the treatment with the necessary perseverance: as soon as considerable amendment is obvious, patients are apt to believe themselves free from their infirmity, and, disregarding advice, to abandon the use of the remedy. Very soon, there is a recurrence of the incontinence, whereupon it becomes necessary to recommence the treatment, and to continue it for a longer time than would have been requisite had the original instructions been strictly followed.

Here, Gentlemen, is a summary of my rules for carrying out this treatment. I order the patient to take every night at bedtime, one centigramme of the extract of belladonna, or half a milligramme [$\frac{1}{110}$th of a grain] of the neutral sulphate of atropine, which may be administered in pills, or in any other form. If the manifestations of incontinence diminish in frequency under the influence of this dose, I continue the remedy in the same dose for a certain period; but if after eight or ten days, the amendment does not progress, I increase the dose to two centigrammes of the extract. I make no change in the time of administering the remedy. Following the same rule, and guided by the same indications, I progressively increase the dose, till I get it up to twenty centigrammes or even more, according to the more or less decided character of the therapeutic action obtained, and the greater or less tolerance for the drug shown by the individual.

When the amelioration has lasted long enough to justify me in believing that the cure is radical—when for three, four, or five months there has been no recurrence of the incontinence—I do not abruptly discontinue the treatment, but I gradually diminish the dose during from two to ten months or longer, according to the circumstances of the case, which may be one of old standing, and consequently more inveterate.

Belladonna administered according to these rules, and administered with great perseverance is, I say again, the most powerful therapeutic weapon with which to oppose nocturnal incontinence of

urine depending upon excessive irritability of the bladder. In these cases, the belladonna acts by diminishing this excitability, this augmented tonicity of the muscular coat of the bladder: it acts, therefore, in virtue of its physiological properties, which consist in diminishing the contractility of the vesical muscular fibres. These properties have been principally elucidated by the experiments which Dr. Comaille made upon himself.

While I reiterate the necessity of gradually augmenting the dose of this remedy, and continuing to give it long after the incontinence has ceased to manifest itself, I must tell you that frequently when the belladonna has produced the desired effects during the first months of the treatment, it is a good plan to suspend its use, administering in place of it for a time preparations of *nux vomica*.

When nocturnal incontinence of urine does not depend exclusively upon an excess of irritability of the muscular fibres of the bladder, but also on atony of the sphincter, belladonna does not prove so beneficial. In some such cases, there is at first a beneficial effect produced, by its lessening the tonicity of the muscular coat of the bladder when that tonicity is in excess, and so facilitating the resistance of the sphincter; but it is generally against the atony of the sphincter that we have to contend, to arrive at the desired result. Now these are the conditions under which the preparations of *strychnine* are indicated. The preparation to which, on account of convenience in prescribing, I give the preference, is the syrup of the neutral sulphate: on former occasions, I have given you the formula for making it, and have also pointed out the mode in which it ought to be administered.

This is the medicine to which recourse ought to be had at once, when the incontinence is exclusively the result of atony, not merely of the sphincter, but of the entire bladder. This kind of incontinence is recognised by its being both diurnal and nocturnal; and also, by the patients passing their water in a weak stream, while the very opposite is observed in those whose incontinence is exclusively nocturnal.

I prescribed strychnine for our patient of bed 1, St. Agnes's ward; but although it soon modified the paralysis, so that the patient was able to walk much more easily, and for a longer time than before he began to take it, it produced no effect upon the incontinence of urine. Seeing this, I entertained the idea of having recourse to *compression of the prostate*, which I have already brought under

your notice as one of the most powerful means which we possess of curing some forms of spermatorrhœa. I believed that by the aid of the compressor of the prostate, I should be able to act on the neck of the bladder, just as in cases of involuntary seminal emissions, I had acted on the orifice of the ejaculatory canal. I gained my object; for the patient was forthwith apprised of his want to urinate, and so was able, which had not previously been the case, to retain his urine when asleep as well as when awake. Although this is the only case of the kind which I can report to you, it is not on that account the less valuable. I advise you to make a note of it.

In addition to the medical and surgical treatment of incontinence of urine, there is another means which I ought to mention; and which consists in impressing upon the patients the importance, during the day, of resisting as long as possible the want to urinate.

Nocturnal incontinence of urine, as I have already stated, depends upon an excess of tonicity and irritability in the muscular fibres of the bladder. Now, whenever a muscle is in this state of tonicity, of abnormal irritability, tending to spasm, the best means of combating that spasmodic tendency is to combat the tonicity of the muscle. To accomplish that object, it is sufficient to carry extension of the muscle to the utmost degree which is practicable: now, it is by habit, that this degree of extension is obtained.

You all know that great eaters have at last larger stomachs than other persons. This increased amplitude of stomach, primarily occasioned by the presence of too large a quantity of food, becomes at last permanent. The muscular coat, from being constantly distended, loses its tonicity; and consequently, the time comes, when the stomach, even though empty, no longer contracts upon itself. In the same way, habitual constipation leads to abnormal distension of the large intestine. This excessive distension of the stomach and intestine is, let me remark in passing, the cause of flatulence and other symptoms regarding which I shall have to speak to you at length on a future occasion.

What takes place in the digestive canal, takes place in the bladder, as well as in all the organs which are hollow: the muscular fibres which enter into the composition of these organs lose in part their contractility by being habitually distended. This is the fact which has to be applied to the special case now before us.

Everybody knows that the bladder is larger in women than in men: this is partly because it is larger naturally in women, but

undoubtedly it is also partly owing to women early acquiring (under the pressure of the social conditions in which they are placed) a habit of retaining the urine for a much longer time than is necessary for men. Some men, however, can acquire a similar habit, and remain many hours without relieving the bladder, while others have no power to resist the first sensation of a want to make water. After a certain age, there may be some objections to retaining the urine for a long time, but none exist in youth, and still less are there any in childhood. We cannot, therefore, too strongly advise children and adults affected with nocturnal incontinence, to retain their water as long as possible when awake. Perhaps to some men of science, this little precaution may seem insignificant: it has, however, not the less its importance, and is an auxiliary to the treatment which I have indicated.

LECTURE LXIV.

GLUCOSURIA: SACCHARINE DIABETES.

Presence of Sugar in the Urine not sufficient to constitute Diabetes.—Transient Glucosuria.—Glucosuria Symptomatic of Cerebral Affections.—Alternating Glucosuria in Gouty Persons.—Persistent Saccharine Diabetes.—May in the first instance be Intermittent.—Symptoms.—Polyuria.—Character of the Urine.—Excessive Thirst.—Period of Wasting: it may be First Period.—Phthisis.—Spontaneous Gangrene.—Intercurrent Diseases and a Febrile Condition suspend Glucosuria.—Pathological Physiology of Glucosuria.—Treatment.—Diet the most important part of Treatment.

Gentlemen:—I have to speak to you to-day of a patient admitted some days ago to the clinical wards: he occupies bed 16 St. Agnes's ward. He suffers from saccharine diabetes.

This man is 36 years of age, and apparently is of robust constitution. However, upon interrogating him as to family antecedents, I learned that two of his brothers had died from disease of the lungs. Although we found in him no sign of thoracic disease, it was incumbent to take into account this piece of family history, as it gave ground for fearing that, in virtue of the hereditary predisposition, pulmonary phthisis, a complication so frequent in saccharine diabetes, might set in sooner than in other diabetic patients. The patient told me that he had been subject habitually to profuse sweating in the hands and feet: but that these sweats had completely ceased from the time of the appearance of the first symptoms of the affection which brought him to the hospital. He maintains, however, that his present symptoms did not come on in connection with a sudden suppression of the customary sweating, but on the contrary that the sweating ceased after the appearance of the diabetic symp-

toms, this appearance having been sudden, which is unusual. Likewise, all at once, the disease assumed that particular form which has been called *phthisuria*, being that form of phthisis in which there is excess in the quantity of the urinary secretion; and which is characterised by the phenomena of consumption, which, as a general rule, do not show themselves in diabetes till the last stage of the disease.

The patient states that last year, in the month of June, consequently about nine months ago, he was mowing in the meadows, the weather being exceedingly hot. To appease a devouring thirst, he swallowed an enormous quantity of milk and water on returning home. From that day, his health, which till then had been unexceptionable, underwent obvious deterioration. He became tormented by unquenchable thirst, and at the same time completely lost his appetite, so that for a fortnight he had no relish for food. This, Gentlemen, is a peculiarity in the case which you must note, for it is opposed to the general rule, an excessive appetite being usually met with in diabetes.

The patient naturally disposed to take alarm, observed himself with the greatest care, and entered into the minutest details with me. He told me, that taking fright at his want of appetite, he had had himself weighed from time to time, and that he found he had lost some kilogrammes in weight. He had also remarked, that when he made water in his garden, an unusual looking trace was left on the ground and grass; and that the bees alighted and settled on the place he wet, to derive from it the juices which they usually rob from the corollæ of flowers.

Uneasy as to his condition, which was becoming worse every day, he sought admission to the hospital at Rheims, where he was under the care of the late Dr. Landouzy, one of our worthiest and most regretted provincial brethren. Dr. Landouzy subjected him to a treatment in which alcaline drinks played the most important part, and under the influence of which the symptoms improved so quickly that he requested to be allowed to return home. His thirst became less urgent, and his plumpness returned. However, he was soon obliged to return to the hospital; but he again left it, having again notably improved. As formerly, his amendment was of short duration: in despair, he resolved to seek farther advice, and with that view came to Paris to seek a cure at my hands, but this I could not promise him.

His glucosuria is of a bad kind, against which medical treatment

cannot prevail. I can only like my honorable colleague at Rheims check the symptoms; and like him, I can only temporarily arrest them. Whatever I do, the disease will resist my efforts; and if this man agree to remain with us, you will see the extreme symptoms of consumption supervene, and ultimately prove fatal.

Gentlemen, do not lose sight of this man, for it is not often that you have an opportunity of studying saccharine diabetes in our hospitals. This does not arise from the rarity of the disease: on the contrary, as has been remarked by Copland, by Graves (of Dublin), and by his countryman Sir Henry Marsh, glucosuria is common—much more common than is generally supposed. In many cases, it escapes observation, in consequence of its not producing any great disturbance of the system, the patients pursuing their usual occupations without seeking medical aid. When diabetic patients come to consult a physician, it is often for anomalous symptoms, the signification of which they do not know, and which do not excite any suspicion in their minds of the possibility of their being diabetic.

Sir Henry Marsh, in the course of his researches into the nature of this disease, interrogated, upon this point, all the patients whom he thought might have diabetes; and he found it in many of them who only complained of dyspeptic or nervous symptoms. In my own clients, as well as in the patients of other physicians to whom I have had the honour to be called in consultation, chemical analysis has revealed glucosuria in the urine of persons, who, to all appearance, had no serious symptoms, and were apparently in the enjoyment of excellent health.

Let me add, however, that since the publication of the researches of modern observers have called attention to the disease, it has been much less common than formerly for glucosuria to pass unnoticed. In some cases, the symptoms as stated by the patients have no significative character; but amid the different symptoms which they mention, we can generally lay hold of some signs of great value, by which we are put on the way to a correct diagnosis, which we complete by examining the urine and finding that it contains glucose.

However, Gentlemen, before laying before you the remarks which I have to make on saccharine diabetes, there is a point on which we must have no misunderstanding; viz., *that the presence of sugar in the urine is not enough to constitute the disease called saccharine*

diabetes, any more than the presence of albumen in the urine is enough to constitute Bright's disease.

Our great physiologist, Claude Bernard, to whom medicine is indebted for the most exact ideas we possess regarding the pathogeny of diabetes, has taught us that sugar appears temporarily in the urine under a great number of different conditions of the economy. This transient, temporary *glucosuria* may supervene from the ingestion of particular articles of food or medicine, such as ether for example, which seem to act by imparting an augmented activity to the glucogenic function of the liver. In these cases, it is true, the sugar appears only in small quantity. There are other cases, however, in which it appears in greater abundance, as in persons under the influence of violent emotions and moral impressions. This *acute glucosuria*, to adopt the name given to the affection by the eminent professor of the College of France, ceases spontaneously and quickly.[1]

The appearance of sugar in the urine of hysterical and epileptic patients, which has been pointed out by Drs. Michéa and Alvaro Reynoso, as well as by others, is likewise referable to a temporary disturbance of the nervous system. The fact, however, I must state, has not been confirmed by all practitioners, although it is given by the physicians whom I have named, as the result of a great many minutely reported cases.

This accidental glucosuria may be also the consequence of an affection seriously implicating the nervous centres. It will be transient or permanent, according to the transient or permanent character of the affection on which it depends.

Goolden, Istrighson, Paggle, and others, have reported cases of temporary glucosuria supervening consecutive to concussion of the encephalon from falls or blows on the head.

From the examples which I could cite, and some of which are detailed in the memoir published by Dr. Fischer,[2] I shall recapitulate the case given by Dr. Szolskaski (de Savigny-sur-Beaune) in the *Union Médicale* for 23rd April, 1853. The patient became glucosuric after a fall, in which he received a fracture of the cranium, with depression of the bone at the middle of the sagittal suture. On

[1] Bernard (Claude):—Leçons de Physiologie Expérimentale Appliquée à la Médecine. Paris: 1853.

[2] Fischer:—*Archives Générales de Médecine*, for September and October, 1862.

the day following the accident, symptoms of diabetes declared themselves. There were urgent thirst, polyuria, and the urine contained glucose. The glucosuria ceased spontaneously at the end of five weeks, at the same date that the other symptoms disappeared.

To such cases ought to be added those originating in falls on the feet, succussions, violent efforts, fractures of the vertebræ, and blows on the back, chest, or limbs. The development of diabetes under such circumstances is explained by the traumatism of the spinal nerves, sympathetic nerve, and spinal marrow—a traumatism acting more or less directly on the ganglionic elements or sympathies which enter into the composition of the cerebro-spinal nervous system, and which are accumulated most abundantly upon the floor of the fourth ventricle, where they terminate; and whence proceed the nerves of organic life which preside over the vaso-motor visceral system.

It is, however, important to recollect, that traumatic glucosuria, though usually transient, sometimes continues for a long time after the cessation of the cause in which it originated. Eight cases of this kind derived from different authors are recorded in Fischer's work.

Gentlemen, A. Becquerel,[1] and before him Dr. Leudet (of Rouen),[2] have described cases of persistent glucosuria symptomatic of serious alterations of the brain; and more recently, Dr. Levrat-Perroton took as the subject of his thesis, sustained before the Faculty of Paris in 1859, *a case of glucosuria* caused by a colloid tumour enclosed within the fourth ventricle.

You have seen the man, between thirty-five and thirty-six years of age, who came into St. Agnes's ward for an attack of polyuria which seemed to date far back. According to the information we received, he had at a previous time passed saccharine urine, but on admission to our wards, his urine was free from sugar. He fell into a state of profound cachexia, and speedily sunk, having had *purpura hemorrhagica* in the last days of his life.

The following is an account of the examination of the brain made by Dr. Luys:—The anterior wall of the fourth ventricle was more vascular than in the normal state: the large venous trunks were

[1] Becquerel:—Etudes Cliniques sur le Diabète et l'Albuminurie. [*Moniteur des Hôpitaux*, 1857.]

[2] Leudet (de Rouen):—Recherches Cliniques sur l'Influence des Maladies Cérébrales sur la production du Diabète Sucré. [*Gazette Médicale de Paris*: 1857.]

delineated on its surface. Moreover, on looking more closely at the brain, some tawny spots were observed disseminated in the upper parts below the superior processes of the cerebellum: there were also observed some other similar spots below the origin of the auditory nerves.

In making a transverse section of the region, Dr. Luys ascertained that the whole of the grey substance was the seat of unusual vascularity, which gave it a pink colour. The histological examination of the tawny spots showed, that these unusual discolorations were due to fatty degeneration of all the nervous cells of the corresponding parts. These nervous cells, in place of presenting regular shapes, fringed prolongations, and circumscribed nuclei, were all transformed into an irregular granular mass exclusively constituted by aggregated yellowish granulations more or less loosely attached to each other. So complete was the alteration of tissue, that it might be said that the histological elements had reached the last stage of retrograde evolution, and had ceased to exist as anatomical individualities.

Dr. Luys had, in the previous year, communicated to the Biological Society the history of a similar case which occurred in his practice. The patient, a man of fifty, diabetic for two years, was seized, in the last period of his existence, with all the symptoms of pulmonary phthisis, under which he sunk. This patient had also double cataract.

At the autopsy, there was found great vascularity with brownish discoloration of the anterior wall of the fourth ventricle, which was also notably attenuated. The histological examination disclosed, a remarkable turgescence of the minute capillaries, and showed that the presence of the yellow patches, in some places tawny and brownish, was solely due to a peculiar degeneration of all the cells of the nervous tissue. All the cells in progress of retrograde evolution were filled with yellowish granulations having jagged, half-destroyed edges, and presenting only some scarcely recognizable fragments.

These clinical facts confirm the results of the remarkable experiments of Claude Bernard upon the production of artificial diabetes, experiments to which I shall have to call your attention when I discuss the pathological physiology of glucosuria.

There are also cases in which temporary glucosuria appears consecutively to irritation affecting directly the liver. Claude Bernard mentions a case in which after a kick from a horse in the right

hypochondrium, the patient had sugar in his urine till he recovered from the accident. Similar cases would perhaps have been more frequently recorded, had the attention of physicians been more directed to the relation between diabetes and diseases of the liver.

When the influence of gout on the liver is considered, it may be asked, whether it is to direct irritation of that organ or to sympathy, that we ought to ascribe the alternations of diabetes which occur, and which sometimes seem to be manifestations of the gouty diathesis, and to succeed in fits the other manifestations of that diathesis.

Claude Bernard states that sometimes gouty patients whose urine contains a great deal of uric acid, suddenly present symptoms of saccharine diabetes; and whose urine is then found to contain a considerable quantity of glucose. He supports this statement by the testimony of Dr. Rayer, who had observed cases of this description: and he says, that he himself had seen a very characteristic case of the same kind.

The glucosuria which is accidental, symptomatic, and transitory, does not constitute, I repeat, the special disease for which the name of "saccharine diabetes" ought to be reserved; and in which, examination of the dead body does not reveal any appreciable lesion which is at all characteristic. Glucosuria, in fact, no more constitutes diabetes, than albuminuria, when symptomatic of disease of the heart or serious fevers, constitutes the special affection which is called "Bright's disease."

This remark does not apply to *intermittent diabetes*, or *periodic diabetes*, both of which perhaps are only different forms of true diabetes. In intermittent diabetes, sugar only appears in the urine during digestion, but this form of the affection often at last becomes continuous diabetes: and periodic differs from intermittent diabetes only in the sugar existing at distinct periods, and at long intervals.

To constitute diabetes then, it is not only necessary that the urine contain glucose, but also, that there exist certain special phenomena the value of which cannot be appreciated, unless specially studied in the patients.

In general, the first thing which strikes one is the great, sometimes unquenchable, thirst, which torments the individuals: sometimes, it is the only symptom of which they complain, and for which they apply to the physician for relief. Its importance is so great, and so universally recognised, that when it is present, the idea that

diabetes exists is immediately suggested to the physician who is consulted, and the same view often also occurs to the patients themselves, whom it exceedingly alarms. It frightens them still more, if the unquenchable thirst be, as it generally is, coincident with more frequent micturition, and with the passing an increased quantity of urine.

The two leading symptoms, then, of saccharine diabetes are:—immoderate thirst, and augmented urinary secretion.

They are not, however, in themselves absolutely decisive as to the nature of the disease; for we meet with both in that kind of non-saccharine diabetes, more appropriately named polyuria or *polydipsia*. We shall even find, that they are perhaps more prominent symptoms in polydipsia than in glucosuria.

If in either of these forms of diabetes, the quantity of urine passed in the twenty-four hours corresponds to the greater quantity of fluid drunk by the patient; if, as generally happens, the quantity of urine greatly exceeds the quantity of liquid taken into the stomach, this excessive urinary secretion is generally much more decided in polydipsia than in glucosuria. It is in cases of polydipsia that from 15 to 100 kilogrammes of urine are passed in the 24 hours.

In saccharine diabetes, the urine often, at first, presents nothing remarkable: when it becomes increased in quantity during the course of the disease, the increase is not so enormous as in the cases I have just referred to; and in the last stage, the quantity is sensibly diminished. Finally, it is not uncommon, as was stated in 1845 by Dr. Contour in his excellent inaugural thesis, for saccharine diabetes to exist, although the urinary secretion be normal in quantity. Moreover, in the same individual, immense variations occur in the quantity of urine secreted under the influence of causes which are very different and often inappreciable.

In most cases, however, more than the normal quantity of urine is passed. During the day, the patients are obliged to empty the bladder much more frequently than is usual with them, and during the night, the want to make water is still greater, obliging them to get up four, five, or six times.

The urine presents decided modifications in its physical properties and chemical composition.

When passed, it is at first transparent, and lighter in colour than the urine of health, being in some cases nearly colourless: when

examined on some particular day, it will be found to have a yellow tint like amber, or to be slightly green: pretty often, it is frothy. Its density is increased; and this increase of density is one of the leading characteristics of glucosuria: from the normal specific gravity of from 1,015 to 1,022 it rises to 1,030, and (according to Bouchardat[1]) even to 1,074; whilst, on the other hand, in polydipsia, it falls to 1,007, to 1,004, or even lower. When allowed to remain at rest for some time, it becomes whitish, resembling clarified whey in appearance, or, to adopt Cullen's comparison, like a mixture of a small quantity of honey with a large quantity of water. Cullen's comparison is all the more applicable that the odour of honey and water resembles that of saccharine urine, which, when it dries on linen, leaves traces similar to those of strongly sugared water. Moreover, we have seen in the case of our patient in St. Agnes's ward—and others have recorded similar facts—that when the urine was spilt on the ground, the flies came to suck up its sugar. Finally, the urine of glucosuric patients, deflects to the right polarised light, a circumstance which has been turned to account in diagnosis.

Saccharine urine, like normal urine, is acid in proportion to its properties and chemical composition; and its acidity is sometimes augmented by the presence of carbonic and acetic acids, the products of its fermentation.

It has long been believed, on the authority of eminent observers, among whom it is enough to mention Thenard, that the urine of diabetic patients contains neither urea nor uric acid. New analyses, however, made by MM. Macgregor, Chevreul, Bouchardat, and others have shown, that there is as much *urea* in the urine of glucosuric patients as in that of persons in good health, and that in both, it is proportionate to the quantity of azotic aliments which they take. *Uric acid*, it is true, is seldom found in saccharine urine, but the presence of sugar is not incompatible, as has been alleged, with the presence of uric acid, as is evident from the latter being in sufficient quantity to deposit crystals.

When albumen is found in the urine of glucosuric patients, it is when they are in the last stage of the disease; it is, therefore, an unfavourable, and not, as Thenard and Dupuytren supposed, a favourable symptom: on this point, clinical observation is in complete accord with the results of physiological experiments.

[1] Bouchardat:—Du Diabète Sucré, ou Glucosurie: Son Traitement Hygiènique. Quarto, Paris, 1851.

The pathognomonic character of glucosuric urine is the presence of more or less sugar, but sugar of a particular kind, *glucose*, which is similar to starch sugar or grape sugar. I shall not enter into the details of the different analytical processes by which its presence is ascertained. You can always easily obtain *caustic potash*, should you be unable to procure more sensitive reagents, such as the solution of Frommherz, of Barreswil, of Fehling, or of Quevenne. On putting a certain quantity of caustic potash into a glass tube containing the urine, or more simply, into a metal spoon, and heating it at the flame of a spirit lamp or candle, the liquid, as soon as it begins to boil, will assume a reddish brown colour should it contain glucose, but this will not be the case in respect of any other urine submitted to the same test.

Besides the intense thirst experienced by the patients, there is another symptom of great value, although it cannot be regarded any more than the other as characteristic of saccharine diabetes, since it is also a symptom of polydipsia, in which indeed it often exists in a more aggravated form : this symptom is an excessive appetite—a real boulimia.

This unnatural appetite is observed in almost all diabetic patients. It may exist to such an extent as to seem an impossibility to satiate them; and it is said that some have eaten in twenty-four hours a mass of alimentary matter equal in weight to the third of the weight of the individual's body.

Notwithstanding the ravenous appetite, and the perfect digestion of the food, nutrition is badly accomplished, the nutritive functions being perverted by the disease; and consequently, diabetic patients rapidly lose flesh, and wasting of the body inevitably leads them to the tomb.

The proposition now enunciated is far too absolute if formulated in this general manner. No doubt, diabetes in its last stage is a consumptive disease: in cases such as that of our patient in St. Bernard's ward, the malady, progressing with excessive rapidity, and passing, so to speak, at once into its second stage, immediately induces great emaciation, justifying its being called *phthisuria* or diabetic phthisis; but generally speaking, this great wasting does not occur in the first stage, which is sometimes very prolonged.

This occurred in the case of a man aged 28, whom you have very recently seen in bed 3 of St. Agnes's ward. Since his admission on the 24th March last, I have been struck with his emaciation and

feverish state. The presence of these two conditions have led me to announce to you unhesitatingly a most unfavourable prognosis, and to predict a speedily fatal termination.

Two years ago, this man was thrown out of work, and reduced to be a deliverer of newspapers. He was thus jaded, and without the means of restoring the waste of the body by adequate alimentation. He soon becamc lean, pale, and at last very weak. Yet, it was not till fivc weeks had elapsed, that he began to feel intense thirst and increased appetite. He had been three weeks in this state at the date of his admission to the hospital: he then had a very marked typhoid appearance: his step was tottering, his look expressionless, his countenance sad, and his tongue dry and rough. He had headache. Eight days previously, he had had epistaxis. His liver was enlarged, and very hard: it extended three finger breadths below the false ribs, and occupied the epigastric region: it was neither hobnailed nor painful. The pulse was 112, small, and not rebounding. The skin was hot and dry.

When admitted to our wards, the patient was drinking from nine to ten litres of fluid daily, and passing a proportionate quantity of urinc. The specific gravity of the urine was from 1,029 to 1,030 in place of 1,015, the natural weight: it reduced, with energetic action, the solution of Frommherz, and assumed a dull red colour, when boiled with caustic potash. The disease was evidently saccharine diabetes.

I prescribed ten grains of levigated chalk to be taken daily; and I ordered at the same time a restorative diet. The treatment, however, produced no improvement. Ten days later, I tried the effect of the inhalation of oxygen, which was continued for five days; and this also was productive of no amelioration. Two days later, on the 11th April, the fever increased, the appetite failed, and the thirst diminished: at this time, the patient was not drinking more than about two litres of fluid a day, and passing a proportionate quantity of urine. His tongue was dry, his debility extreme; and he had fallen into such a state of marasmus as to be unable to leave his bed. He died two days after this in a state of sub-delirium, having been ill only five weeks. During the last three days of his life, the urine was not at all copious, but it nevertheless contained a large quantity of sugar. It is right to add, that from the time this man came into the hospital, I was struck by the almost bronzed appearance of his countenance, and the blackish colour of his penis.

At the autopsy, we did not find any morbid state of the supra-renal capsules. The kidneys were neither more voluminous nor more vascular than natural: they were neither in a state of hyperæmia nor hypertrophy; for from the disease not having been of long duration, and the urine not having been excessive in quantity, they had not suffered from excessive functional action. It was otherwise with the liver, which was at least twice its normal volume: the length of the right lobe was nineteen centimeters: the left lobe, which extended to the spleen, was twenty centimeters in length: and the total length of the liver was thirty-four centimeters. The entire surface of the organ was granular: it was of a uniform greyish yellow colour: it was very dense, resisting pressure so much as to prevent penetration by the finger. It creaked under the scalpel; and the surface of the cut was granular in place of being smooth. There was well-marked cirrhosis; but the cirrhosis was hypertrophic. The morbid change did not involve the fibrous, so much as the secreting tissue. The fibrous capsule, and the trabeculæ which segment the liver, were increased in thickness; and there was a still more decided augmentation of the volume of the acini, which were visible projecting from the surface, and also visible on the surface of the section, to which they gave a granular character. Thus, there was hypertrophy of the liver consequent upon excessive functional activity, hypertrophy specially involving the secreting tissue of the organ. This was evident upon microscopic examination, by the aid of which one could see that the hepatic cells, so far from being destroyed or atrophied, were increased in volume and in number.

Gentlemen, I cannot too earnestly call your attention to this morbid alteration of the texture of the liver, which is completely in accord with the theory of Claude Bernard. The glucose increases within the system from the time that it appears in large quantity in the urine; and the result is hypertrophy of the secreting tissue of the organ, by which the glucose is produced.

Grant that the immediate cause of glucosuria is a particular state of the liver—has it not elsewhere a more remote starting point? I likewise attach special importance to the anatomical state of the floor of the fourth ventricle. Now, I ought to tell you, that there was nothing different visible to the naked eye in the fourth ventricle of this man from the appearance presented by the fourth ventricle in any other subject. The vascularity was not greater, and the colour

was the same. Moreover, microscopic examination did not disclose amid the cells of the nervous tissue, or beneath the lining membrane of the ventricle, the hematic deposits, the granular globules which have been pointed out as occurring in some cases of diabetes.

It appears then, that in these cases, there is complete proof of lesion of the liver, of its being hypertrophied, and in a state of abnormal functional activity, the result being glucosuria.

Gentlemen, you will often see diabetic patients not only not become thin, but actually become fat. A near relation of my own has been affected for six years with saccharine diabetes, accompanied by voracious appetite; and yet, he has manifestly increased in plumpness. There has in this case been no deterioration of general health nor of mental capacity.

Graves relates a similar case. The patient was a gentleman of Dublin who had had glucose in his urine for seven years: his appetite was wonderful, his physical vigour extraordinary, and he continued to direct with great intelligence and activity extensive agricultural concerns which he had in the country.

It has been said, that in consequence of the *cutaneous secretion* being badly performed in diabetic subjects, their skin is dry. As a general rule, this is true; but nevertheless, there are exceptions to the rule. The person, of whom I was speaking a minute ago, generally had his body bathed in perspiration; and Graves mentions patients in whom he had seen copious sweating. Profuse perspiration is sometimes, though very exceptionally, met with even in the second stage of the disease, in which the skin is almost invariably exceedingly dry.

Coincident with this perversion of the cutaneous functions, there is another symptom, one which is met with much more frequently in women than in men: I refer to an eczematous eruption in the private parts, sometimes accompanied by very painful itching. When you are consulted by women who are becoming elderly, for intense itching in and around the vulva—when on examining the parts, you find that there is eczema, and learn that it has come on irrespective of the menstrual periods, or of any leuchorrhœal discharge, and that the pain it occasions is so great as to prevent sleep, the probable existence of glucosuria will suggest itself. You will often be told by the patient that this eruption, apparently an altogether local affection, is coincident with excessive thirst, and profuse discharge of

urine, which secretion on applying the potash test, you will find, contains sugar.

It is not a very unusual occurrence, that saccharine diabetes should manifest its existence only by anomalous nervous symptoms, which are only explained by referring them to this disease, and the nature of which is first revealed by chance leading to the discovery of sugar in the urine. I say that it is frequently by chance that we are led to a correct diagnosis; for when the glucosuria is unaccompanied by polyuria, the quantity of urine passed in the twenty-four hours remains normal, or is very little augmented. There is also very often, I repeat, an absence of the dyspeptic symptoms of which I spoke at the beginning of the lecture, as well as of voracious appetite and excessive thirst.

The anomalous nervous symptoms to which I refer may consist in diminished motor power, or in perverted sensibility. A remarkable case of exalted sensibility occurred in my practice.

The patient was a woman about sixty, who although she retained an appearance of perfect health, had, for three years, complained of feeling constant pains in the whole of the right side. These pains, which the patient compared to twingings and cramps, sometimes aggravated momentarily, allowed her no respite: touching the parts increased the pains, and even the mere contact with her clothes often produced this effect, though somewhat firm pressure on the painful parts occasioned no disagreeable sensation. Notwithstanding this hyperæsthesia, the affected side retained, as perfectly as the other, its motor power and muscular strength.

The general health, I repeat, was otherwise good: all the functions of organic life seemed to be performed with the greatest possible regularity. The lady's appetite remained as good as it ever was, and there was no dyspepsia: latterly, she had had usually a slight degree of constipation. The nervous symptoms had continued three years, but it was only for one year that she had been known to have glucosuria. At this date, and for some days previously, the patient having suffered from somewhat urgent thirst, attention was directed to her urinary secretion, although it was not much more copious than usual. Upon being chemically examined, it was found to contain a considerable amount of sugar. From that time, the proportion of sugar varied much, sometimes entirely disappearing, and at other times, reappearing in large quantity. The nervous symptoms have not undergone any change.

Enfeebled vision, premature presbyopia, is one of the most common, as well as one of the most remarkable of the symptoms of saccharine diabetes, which are referable to the nervous system, symptoms which become more and more prominent as the disease advances.

A man, for example, in the prime of life, tells you that for sometime his sight, hitherto perfect, has obviously deteriorated—that for some time he has been unable to read without placing the book at a greater distance from him than was formerly necessary, and that then spectacles had become requisite—that with each succeeding month he had to use stronger and stronger glasses. From this fact alone, you have reason to think that the patient has either albuminuria or glucosuria. This symptom alone, even when others are absent, will suggest to you the propriety of examining the urine, and by that examination your diagnosis will be cleared up.

This presbyopia, an ordinary symptom in persons affected by saccharine diabetes, increases rapidly: it is observed in the first stage, and being dependent, as I have been telling you, upon disorder of the nervous system, becomes more evident as the disease approaches nearer to its second stage. In some cases, transient or permanent amlyopia is observed in diabetic subjects. It is the result of an organic change in the retina; and is only met with in the second stage of diabetes. At other times, cataract, generally cataract in both eyes, is developed in diabetic subjects, during the latter months of their existence. The reality of diabetic cataract has been established by the researches of Claude Bernard; and the cases have been collected by Dionis, Leudet, and Græfe. In a memoir by Lécorché,[1] you will find an interesting discussion on the nature of these cataracts, and the manner in which they are formed.

During the second, or to speak more correctly, the consumptive stage of diabetes, which in some persons sets in suddenly, the digestive functions become impaired: the appetite is depraved, and the patients have a disgust for food: they suffer from gastric pains, which increase after eating, from nausea, eructation, vomiting, and diarrhœa, which latter after alternating with constipation at last becomes very profuse. The mouth, acid and dry, as in persons suffering from thirst, is hardly moistened by the thick, frothy saliva which forms whitish tracks upon the bright red tongue, on the

[1] Lecorché:—De la Cataracte Diabétique. [*Archives Générales de Médecine* for May, 1861.]

mucous membrane of the cheeks, and on the commissure of the lips. Although there be no sugar in this saliva, diabetic patients often complain of always having a sweet taste in the mouth. This, Claude Bernard explains by comparing the phenomenon to one analogous to it observed in dogs into whose blood-vessels a decoction of meat has been injected: they immediately lick their lips, thereby indicating the existence of an agreeable sensation. He says, that there is ground for believing, that both in the dogs experimented on, and in the diabetic patients, the substance (existing in the blood in large quantity), is carried with the blood into the capillaries of the mucous membrane of the mouth, where it acts on the sentient extremities of the nerves, as if it had just been directly absorbed by that mucous membrane. But though there be no sugar in the saliva of diabetic patients, their sputa contain it: the sputa consist of bronchial mucosity, secreted by the patients frequently in great abundance in consequence of their very often becoming phthisical in the last stages of the disease.

Under these circumstances, they are subject to a dry cough, which seems to be excited by a troublesome tickling in the larynx. This cough soon becomes a source of anxiety; and auscultation, which at first yielded only negative signs, reveals the existence of pulmonary tubercle which passes rapidly through the different stages of evolution.

Then, likewise, the derangement of the nervous system becomes more manifest. The mind becomes affected, and hypochondriasis, which in a few cases appears as one of the first symptoms of the disease, assumes formidable proportions, and attains such a degree as to be insanity. The procreative power, often increased at the beginning of glucosuria, diminishes, and is ultimately entirely lost. Sensations of internal heat, alternating with rigors, and a greater sensitiveness to external cold, concur in giving proof of perturbation of the nervous system, and bad state of the circulatory functions. It is then also, that the patients become the subjects of *albuminuria*.

The impaired condition of the circulatory and nervous systems are still more strongly manifested by those remarkable symptoms to which the attention of the medical world has been directed by Dr. Marchal (de Calvi).[1] I refer to *spontaneous gangrene*, simulating

[1] Marchal (de Calvi):—Recherches sur les Accidents Inflammatoires et Gangréneux Diabètiques—Théorie Nouvelle du Diabète. Paris, 1864.

that affection which has been called *senile gangrene*, which is observed in the course of serious fevers; and regarding which I addressed you at considerable length in my lectures on dothinenteria. In diabetes, this gangrene of the tissues is evidently connected, for the most part, with that condition of the arteries to which the name of *arteritis* has been given. But whether this arteritis precede the formation of the fibrinous plug, or whether, on the contrary, it occur as a consequence of the formation of this clot, (which is the anatomical cause of the gangrene, by its obstructing the circulation of the blood in the parts about to mortify), it is unquestionable, that the symptoms supervene under the influence of a peculiar predisposition, of a general state of the economy, related to the disease which has glucosuria as its characteristic sign.

This spontaneous gangrene chiefly attacks the lower extremities. Let me here give you the details of a case in point, which occurred in a young American lady whom I attended during her residence in Paris. She had left France to return to New Orleans : during the following year, saccharine diabetes, with which she was affected, had not presented any appreciable modification, when all at once, symptoms supervened which rapidly proved fatal.

Her husband sent me the following history of what occurred. It is contained in a long letter, a translation of which, with your permission, I shall now read :—

" Since I saw you, till November last, the state of my wife did not present any material change, but at that date she was greatly prostrated by a choleraic affection. She recruited, however, and, with a wonderful rebound, regained strength and plumpness.

" At that period, acting in accordance with, or rather going somewhat beyond your advice, she abstained from farinaceous food : she entirely discontinued the use of bread. Great improvement was the result. She became strong, and also a little fatter than she was in summer. During the whole winter, she remained free from the itching, and in better health than in the previous year.

" In the beginning of March, a small ulcer, about as large as a pea, appeared on the outside of the fourth toe of the left foot. The foot became inflamed; and with the view of subduing the inflammation and pain, poultices of linseed meal were applied. The limb was kept in a horizontal position.

" About two weeks later, all the bones of the toe were affected and the ulcer extended round the toe, which soon got into such a

state that it became necessary to remove it: this was done without the patient feeling the slightest pain. The pains, however, returned, and became so severe, that it became necessary to use opiates freely; and the question was, whether the patient would have sufficient physical energy to resist the progress of the gangrene. It advanced slowly and insidiously; and before stopping, had nearly reached the instep.

"Such was the state of matters on Thursday, 13th April. The position of the patient was considered critical; but immediate danger was not feared. She passed an exceedingly agitated night, but was strong enough in the morning to be able to rise without assistance. She complained of great heat, of great obstruction in the throat, and of a feeling of being suffocated. Her attempts to make water were very frequent, and it seemed as if micturition was difficult. Between two and four in the morning, her agitation increased exceedingly; and the pulse became very perceptibly weaker. Later in the morning, the agitation ceased, and gave place to tranquillity making semblance of the looked for sleep having come at last: the patient, however, did not sleep, but gradually became weaker, and died about ten o'clock without a struggle, passing away as softly as a child falls asleep."

This spontaneous gangrene occurs sometimes in other parts of the body, as in the chin, nose, continuity of the limbs, and in the walls of the chest: there are even examples of it affecting the lungs.

The case, Gentlemen, which I have just related shows how death occurs in glucosuria; but it is very unusual for the disease to terminate in this mode.

As a general rule, diabetic patients sink slowly, succumbing under the complication of tubercular pulmonary phthisis.

In other cases, the patients are carried off by cerebral apoplexy.

In 1846, when doing duty at the Necker Hospital, I had under my care a woman admitted with saccharine diabetes: the disease was presenting nothing unusual in its progress, when, fifteen days before death, the patient was seized with acute catarrh accompanied by high fever. *From that time, the urine ceased to contain glucose.* Eleven days after the commencement of this bronchitic attack, very violent otalgia of the left ear supervened; and next day, we found that there was hemiplegia of the right side, with complete paralysis of the limbs, accompanied by slight muscular contraction, and some diminution of the cutaneous sensibility. The face did not seem to partici-

pate in the paralysis. The patient fell into a state of stupor and died.

At the autopsy, we found small softened masses seemingly infiltrated with blood, situated in the corpus striatum, optic thalamus, and some parts of the grey substance near the circumference of the left hemisphere of the brain. The meninges seemed to be in a healthy state; and there was no appearance of any lesion of the dura mater in the situation of the petrous portion of the left temporal bone.

In the lungs, there were tubercular masses in the second stage, and some cicatrised cavities.

The kidneys, though hypertrophied and much injected with blood, were not softened; but in the situation of the cleft, there was more swelling, a deeper red colour, and more infiltration.

In my notes of the case, no mention is made of the state in which the liver was found. At the date when the memoranda were taken, the part which the liver plays in saccharine diabetes was not known.

Since the beautiful researches of Virchow have more specially directed the attention of pathologists to the subject of arterial obliteration and the part which it plays,[1] I have often asked myself whether these local gangrenes of which I was speaking a little while ago, whether the cerebral and renal affections which occurred in the case of which I have summarily given you the history, were not caused by real embolia occupying either small arterial branches or more considerable trunks. I am aware that our illustrious brother of Berlin would entertain very little doubt on that point; but many years, probably, will elapse, before these opinions acquire citizenship in our country, or even in that of M. Virchow. It will still be an open question, whether the arterial obliteration is effected *in situ* by the same pathological process which causes the local gangrene, or whether the gangrene is the consequence of obliteration produced by a migratory clot, or by a local morbid change taking place in the vessel.

In the case, with an abstract of which I have just presented you, I laid stress on the fact, that from the time the patient was under the influence of the acute affection, the urine ceased to contain glucose.

Gentlemen, it is a remark of all observers, that when an acute

[1] Virchow:—Gesammelte Abhandlungen. Berlin: 1862.

disease supervenes in diabetes, sugar no longer appears in the urine, so that one might suppose the patient was cured of the diabetes. Now, Claude Bernard has proved by his experiments that perfect activity of the digestive function is the primary essential condition requisite for the liver secreting sugar, and that any deterioration in the performance of that function, be the cause what it may, so long as it lasts, puts a stop to the diabetes.

Though fever is one of the causes, it is not the only cause of disturbance of the hepatic functions; and some other active pathological condition may produce a similar effect, the patient, during a certain period, not presenting the characteristic sign of diabetes.

In relation to this point, Claude Bernard gives the history of an individual who had diabetes in a very obstinate form. For the first few days, it diminished under the influence of remedies, but tolerance for the remedies was then acquired, and the disease returned with its former intensity.

"In these cases," says the illustrious physiologist, "what takes place is quite natural: by each new medication, the functions are disturbed, including those of the liver, and the production of sugar is arrested, but only to be resumed as actively as ever. We must never, therefore, be deceived by such results, nor consider a patient as cured, in whom we have, by the administration of any medicine whatever, temporarily prevented the appearance of sugar in the urine."

The physician when he is called in to cases of saccharine diabetes ought always to have in his mind the judicious remarks which I have now quoted.

Gentlemen, before discussing the question of treatment (the most important part of our subject), it is necessary to enter into some considerations relating to the *pathological physiology of glucosuria*.

Rollo, one of the first writers on diabetes, believed that it originated in impaired digestion, in a derangement of the functions of the stomach, the secretions of which acquired, he supposed, the morbid power of transforming into sugar the alimentary substances which it received.

At the beginning of this century, Nicolas and Gueudeville regarded the disease (which they called saccharine phthisuria) as the result of an affection of the intestines. In their opinion, the chyle, in consequence of a morbid change in the intestinal secretions, in place of being composed of nitrogenous materials consists of less per-

fectly elaborated elements, of saccharine matter, not suitable for the complete nutrition of the body.

These theories, you perceive, differ little from that which has been advanced in our own day by a chemist, M. Bouchardat.

Cullen—who for a short time had adopted the opinion of Mead, that diabetes is dependent upon a certain state of the bile, an opinion founded on the fact that the disease is sometimes met with in persons suffering from an affection of the liver—Cullen seemed nearer the truth, when, abandoning that view, he returned to the idea (too vague no doubt) that the immediate cause of diabetes is a defect in the assimilating function, that function by which the food is converted into proper nutrient fluids. He makes out, however, but a weak case for his theory, for he adds :—"I formerly communicated this idea to Dr. Dobson, who adopted it, and published it; but I must confess that the theory is beset with difficulties, which cannot at present be solved."

It was not till our own time, that the problem was approximatively solved; and the pathogenesis of diabetes still remains, and probably must remain for a long time enveloped in much obscurity.

The science and art of medicine are indebted for the more precise knowledge now existing regarding the disease to the researches of that eminent physiologist whose name is so to speak linked with it. These researches would possess a real unquestionable value, had their only result been the overthrow of the theory of the chemists, who judging of what takes place in the living organism by the results of experiments in their laboratories, were assuming the direction of the therapeutics of diabetes. Had Claude Bernard done nothing more than upset the chemical theories of diabetes, he would have rendered an immense and never-to-be-forgotten service to medicine.

A fundamental fact was at once brought to light. It had always been believed that the proximate principles found in the animal economy were derived exclusively from the vegetable kingdom, which it was supposed had alone the power of producing them, animals only extracting and assimilating them, so as to destroy them. In respect of sugar, in particular, when found in an animal, it was said that it had been introduced with the food; and it was consequently argued, that the quantity of that sugar must vary with the nature of the aliment. Now, as it was also believed, that the saccharine substances, or the feculent substances which are transformed into sugar under the influence of the digestive juices can alone supply it

for intestinal absorption, it was again concluded, that it ought to be found in herbivorous animals fed on feculent substances, but that its presence could not be expected in carnivorous animals fed only on nitrogenous or fatty matters, which, it was said, could not be transformed into sugar by the known digestive processes.

Claude Bernard has demonstrated the inaccuracy of these views. He has proved that sugar is found in all animals, and in equal proportions in the different species in the animal series irrespective of the nature of their food. Latterly, G. Colin has shown by numerous experiments, detailed in a memoir read to the Academy of Medicine, that even nitrogenous substances can be transformed into sugar by the process of digestion.

Claude Bernard, however, has shown that the production of sugar in the animal economy is not only irrespective of the nature of the food, but takes place even independently of alimentation, sugar having been detected in the blood of animals who have not lived an extra-uterine life—in the fœtus of birds, as well as in the fœtus of the mammalia.

If food be one source of the sugar, it is evident that it has likewise another. What is that other source?

I could not, Gentlemen, without transgressing the limits of my teaching in this place, enter into the details of this important physiological question: I must, therefore, send you to the works of the eminent professor of the College of France for the full development of this important subject. I shall, on the present occasion, confine myself to a recapitulation of the facts which more immediately bear upon glucosuria.

As soon as it was discovered that the sugar in animals does not come only from the food, it became evident that an organ must produce it, at least in part: it became evident also, that there must be a special function by which the elaboration of saccharine matter is effected; and then came the question:—What apparatus is charged with the performance of this function?

In searching in the different tissues and organs for this saccharine matter, which analysis has shown to be similar to glucose (the sugar of starch), or, to express the fact more correctly, to the sugar found in the urine of diabetic patients (which differs a little from the sugar of starch), Claude Bernard was struck by the circumstance, that in whatever kind of animal he experimented on, the liver was the only organ impregnated with it. From this, he concluded, that this gland

like other glands ought naturally to be impregnated with the products of its secretion, just as the kidneys are impregnated with urine, the testicles with spermatic fluid, the pancreas with pancreatic juice, and the salivary glands with saliva. Sugar was also found in the liver of persons suddenly cut off by death when in a state of health. In the liver then, in addition to the bile—the only secretion with which the liver was formerly supposed to be charged—there is elaborated the saccharine matter found in animals which (like the fœtus) cannot have derived it from aliment, at one time supposed to be its only source.

Claude Bernard, in pursuing his researches, attained demonstrative evidence the most unquestionable, that the liver performed this special office of secreting sugar. On analysing the blood which comes to the liver from the intestines by the vena porta, and that which leaves the liver by the hepatic veins on its way to the vena cava, he discovered that the blood of the vena porta did not contain a trace of sugar, while that of the hepatic veins contained it in large quantity: again, he found that the blood of the vena cava, of the right auricle of the heart, and of the pulmonary artery, contained sugar in quantities progressively diminishing with increase of distance from the liver. Here, then, was an absolute solution of the problem: the saccharine matter was evidently formed in the liver, where it was met with in greatest abundance.

Thus, then, the liver performs two functions; one being the secretion of bile, the products of which, poured into the digestive canal, are subservient to intestinal digestion; the other being the secretion of sugar, the products of which, not excreted like the former, re-enter the general circulation, or at least that part of the circulation extending from the liver to the heart and from the heart to the lungs.

This double function, performed by a single organ in the higher classes of animals, is quite distinct in molluscs, and is still more separate in insects, which have an organ for the elaboration of bile and another for the elaboration of sugar.

I told you, Gentlemen, that the saccharine matter secreted by the liver returns into the general circulation, or at least into that portion of the general circulation between the liver and the heart and between the heart and the lungs. Restricting still more, at this point, the limits of my subject, I now reach the more direct applications of physiology to the pathology of diabetes.

Claude Bernard found that upon searching for sugar in animals killed at different periods of digestion, he discovered that in animals killed at an interval of some hours after their last repast, sugar existed only in the tissue of the liver, and in the vessels going from the liver to the lungs. What then had become of the sugar? As none was found in the blood which had traversed the lungs, it must have been destroyed in them, or before it reached them. How is this destruction accomplished? The theories invented to explain this are not satisfactory; and we are consequently obliged to confine ourselves to a statement of the fact, without endeavouring to explain it. Be the explanation what it may, the phenomenon is one of so much importance, that when it ceases, life ceases: what we do know of it, however, is sufficient so far as the question of glucosuria is concerned.

I have just told you, that when the animal is killed fasting, the sugar secreted by the liver is not found in the blood which has traversed the lungs. It is otherwise, however, when the sugar is searched for two or three hours after a repast. At that stage of digestion, sugar may be found in all the arteries and veins of the body: it is found at that stage, even in the renal arteries, in too minute a quantity it is true, to traverse the kidneys, so that no trace of it is contained in the urine, any more than in the other secretions. It may happen, however, that the secretion of sugar is increased to such a degree that some of it passes off in the urine. However that may be, this sort of saccharine overflow beyond the lungs—whether it be or be not greater than natural—continues about three or four hours, after which the sugar is only found in situations short of the lungs.

From these experiments, it results, that the secretion of sugar in the liver presents variations like all the other secretions; and this primary fact will account for what is observed in diabetic patients, whose urine is more or less charged with glucose, according to the period of the day at which it is passed, whether nearer or more distant from the time of meals.

These oscillations in the quantity of sugar in the blood at the different stages of digestion would seem to indicate, that although the nature of the alimentary substances may not exert an influence upon the production of sugar, alimentation is at least the source whence the organism derives the saccharine matter. Claude Bernard, however, has demonstrated that the liver is the sole source of the sugar. I cannot, I repeat, enter into the details of this question, and must,

therefore, refer you to the lectures which our illustrious physiologist has devoted to the subject. He will tell you that neither sugar introduced into the intestinal canal nor glucose coming from the reaction of the digestive juices upon the feculent substances augment the quantity of sugar in the liver nor in the vessels leading from it; farther, that the sugar and alimentary glucose are destroyed by the liver, and are therein transformed into a peculiar emulsive substance. Recollect this fact: it is of importance that you know it as a guide to the treatment of diabetic subjects.

Gentlemen, I told you that under certain circumstances, sugar passes by the urine, and that when this occurs, it is the sugar of diabetes, that is to say, sugar, which, secreted by the liver, has passed through the lungs before reaching the great circulation. Animals which after having fasted for a long time, eat saccharine or feculent substances, pass urine containing sugar directly derived from these substances. The extreme rapidity with which intestinal absorption takes place in animals after fasting, explains this phenomenon. In consequence of this rapidity, the sugar contained in the absorbed fluids is carried in mass towards the liver. There, a part of this sugar, passing into the vessels belonging to what Claude Bernard calls the chemical circulation of the liver [*circulation chimique du foie*] is destroyed: the other part is carried into the torrent of that other circulation which he calls the mechanical circulation of the liver [*circulation mécanique du foie*]. This collateral circulation, much more rapid than the former, has as its apparatus the vessels of the portal system, which, in place of being imbedded in the lobules of the liver, circumscribe them, and anastomose with the hepatic veins which pour their contents into the inferior vena cava. The sugar, thus poured in greater or less quantity into the general circulation, passes into the urine, where its presence may be detected for a longer or shorter period. Its presence there, however, is only transient, and is a phenomenon in no way peculiar; for a similar result is observed in respect of all alimentary substances taken in too large quantities. Thus, for example, Claude Bernard in his *Leçons de Physiologie*, mentions a healthy man, who became temporarily affected with albuminuria from swallowing a large number of raw eggs after a long fast. It was observed that some hours after this repast of eggs, the individual's urine became very albuminous; and a certain time elapsed before it regained its normal character.

Though digestion has a remarkable influence, not only on that

form of glucosuria in which the sugar is directly derived from the ingesta, and also on the oscillations in the quantity of sugar in the urine and secreted by the liver, this influence (probably depending upon activity in the function of hepatic secretion), is not the only influence in operation.

From the moment that sugar is secreted by the liver, every cause which increases or diminishes the secreting function of that organ will also be likely to increase or lessen the quantity of the sugar in the organism.

Here it is, Gentlemen, that the beautiful experiments of Claude Bernard, in connection with the production of artificial diabetes, have thrown such a flood of light upon this important question of glucogenesis and glucosuria.

Like all other glands, the liver is under the dominion of the nervous system. Acting on the latter, we may act indirectly on the former, in such a way as to stimulate, modify, diminish, or even annihilate entirely the function with the performance of which it is charged.

Claude Bernard says that, if you prick the medulla oblongata of an herbivorous or carnivorous animal in a certain situation within the fourth ventricle (limited superiorly, by a transverse line uniting the two tubercles of Wenzel, and inferiorly, by another line extending between the origin of the two pneumogastric nerves), after some time, sugar will exist in large quantity throughout the body, and appear in the urine. The irritation excited by pricking the nervous centres is transmitted to the liver by the spinal cord and the branches of the great sympathetic which preside over the functions of the liver, the secretion of sugar is augmented, and the blood, saturated with it, carries it to the lungs in so large a quantity that part passes through them without being destroyed, and reaches the general circulation, whence it is eliminated by the stomach (as we infer by its being found there mingled with the gastric juice), and eliminated to a still greater extent by the kidneys, which accounts for its presence in the urine.

On the other hand, if you divide the pneumo-gastric nerves in the neck, or divide the spinal cord above the origin of the branches of the great sympathetic which go to the liver, the secretion of sugar is arrested.

You see then, Gentlemen, that experimental physiology explains the existence of glucosuria in those cases in which it is connected

with lesions of the fourth ventricle of the brain, cases similar to those of which I have given you examples.

Experimental physiology also explains what occurs in cases similar to those reported in Dr. Fischer's memoir, cases in which saccharine diabetes was recognised as the starting point of more or less serious affections of the encephalon, spinal cord, spinal nerves, and great sympathetic. This is the place to lay before you the beautiful experiments undertaken by a very eminent German physiologist, Dr. Schiff, for the purpose of confirming and testing the experiments and conclusions of Claude Bernard: they throw fresh light on the question which now engages our attention.

Since the date of the celebrated experiment of Claude Bernard, it has been known, that glucosuria is produced by pricking the medulla oblongata in the central part of the floor of the fourth ventricle, between the origins of the auditory nerve and the par vagum.

The presence of sugar in the urine results from an excess of sugar in the blood. Now, in reference to this point, Schiff proposed to himself the following problems, which he solved with his usual sagacity.

1. Does the excess of sugar in the blood depend upon there being an abnormally less rapid destruction of sugar in the organism, or upon there being an increased production of it?

2. Adopting either hypothesis:—Does pricking the floor of the fourth ventricle act by paralysing or by exciting the medulla oblongata?

3. How is the nervous action, whether paralysing or exciting, transmitted to the organs which directly produce glucosuria?

Schiff proved that excess of sugar in the blood is really the result of a modification of the function of the liver, by extirpating the livers of frogs, examining their blood three weeks after the extirpation, and finding that it then contained no sugar. It is very evident from this experiment, that simple pricking of the floor of the fourth ventricle is insufficient to produce glucosuria, that the intervention of the liver is absolutely necessary, and, in fact, that it is the organ by which the sugar is formed.

Schiff likewise confirmed the views of Claude Bernard, by tying portions of the liver in frogs previously rendered glucosuric. He found that by this proceeding the quantity of sugar in the urine diminished proportionately to the artificial diminution of the volume of the liver.

Does diabetes arise from an excessive production of sugar by the liver? or, does it arise in consequence of the non-formation of the ferment [*le ferment*], which, under normal conditions, ought to destroy the sugar contained in the blood?

Schiff, with a view to decide in favour of one or other of these hypotheses, tied larger and larger portions of the liver in frogs which had been made glucosuric. It is very evident that if diabetes is dependent upon the non-formation of the sugar-destroying ferment, a time will of necessity come, when, notwithstanding the partial destruction of the liver, notwithstanding the exceedingly minute quantity of sugar produced by the small remnant of liver, the sugar (being no longer destroyed) will accumulate in the blood, and glucosuria will appear—the frogs will again become glucosuric. Now, the sugar diminished in the urine of these frogs, in proportion as the volume of the liver diminished. Hence it follows, that the cause of diabetes is increased production of sugar by the liver, and not non-formation of that ferment which is destined to destroy the sugar.

The solution of the first question then is, that "glucosuria is the result of an excess in the production of sugar in the organism."

But what is the *nature of the change in the nervous system* which gives rise to glucosuria?

Andral pointed out, more than twelve years ago, that there was hyperæmia of the liver in diabetes. Is this hyperæmia the immediate cause of diabetes? and if so, how is the fact to be proved by experiment?

To produce hyperæmia of the liver, Schiff availed himself of an anatomical peculiarity in the frog. In the frog, the liver does not receive the whole of the venous blood of the abdomen: only a portion of it is carried to the organ by the hepatic vena cava, while another portion is conveyed directly to the heart by a second vena cava without passing through the liver. To produce hyperæmia of the liver, it is sufficient to force all the venous blood of the abdomen to traverse the liver; and that can be accomplished by tying the second vena cava. Two hours after the application of the ligature, the frogs were glucosuric.

A similar result follows removal of the spleen; this proceeding causes immediate hyperæmia of the liver, and rapidly consequent glucosuria. Likewise, when traumatic hyperæmia is produced by pricking the liver, the result is glucosuria. But does pricking the

floor of the fourth ventricle produce glucosuria by causing hyperæmia of the liver?

According to Schiff, this pricking irritates the vaso-motory nerves of the liver, which causes dilatation of the vessels of the organ; and as a primary consequence, hyperæmia; and as a secondary consequence, glucosuria. This is in accord with Claude Bernard's theory as to the regulating influence which he attributes to the great sympathetic. The explanation given by Schiff rests upon his belief, that the vaso-motory nerves which regulate the contractions of the vessels belonging to the abdominal organs arise from the optic thalamus and crura cerebri, are united in the medulla oblongata, where they are side by side with the other vaso-motory nerves of the body, then descend by the antero-lateral columns, diverge from one another, finally leave the medulla, traverse the ganglia of the spinal cord, and terminate upon the vessels of the abdominal organs. Supposing this anatomical view to be correct, pricking these nerves at their origin, as they pass in the vicinity of the fourth ventricle, would produce diabetes. Thus, pricking the fourth ventricle will lead to no special result; but any lesion involving the vaso-motory nerves from their origin in the crura cerebri to their termination in the abdominal organs, will produce glucosuria: the floor of the fourth ventricle is, however, the most convenient locality on which to operate, as the vaso-motory nerves are there concentrated within narrow limits. Thus, direct irritation of the vaso-motory nerves of the liver by means of galvanism produces glucosuria: a similar result follows when frogs are poisoned with strychnine or opium, and likewise succeeds a prolonged tetanic state.

If, on the contrary, we were to cut through the anterior columns of the medulla, by which are transmitted the branches of the sympathetic which go to the abdominal viscera, irritation could not be transmitted to the liver, and consequently pricking the fourth ventricle would not produce glucosuria.

It, therefore, follows, that pricking the fourth ventricle produces glucosuria by inducing hyperæmia: that this hyperæmia arises from irritation of the vaso-motory nerves of the liver: that the irritation, again, results from pricking the fourth ventricle, because these nerves are at this point near their origin, collected together; and finally, the irritation is transmitted to the liver by the anterior columns of the medulla.

Thus we arrive at the solution of the second and third problems proposed by Schiff; and which I stated a few minutes ago.[1]

Glucosuria produced by pricking the fourth ventricle is considered by Schiff to be a special form of the affection; and he calls it *irritative* glucosuria. It has an essentially fleeting character, for it does not continue more than some hours, or a day. It is subject to the general law, that irritant action is rapidly exhausted: the irritation is soon deadened, and quickly becomes extinct.

Paralysis, on the other hand, has durable effects: glucosuria of paralytic origin is likely to be permanent. It is this form of glucosuria which Schiff says he discovered. He produced *paralytic* glucosuria by dividing the anterior columns of the spinal cord, because that operation necessarily implied section of the bundles of vaso-motory nerves which traverse them. The section ought to be made at a point corresponding to the fourth cervical vertebra, or nearer the medulla oblongata. The vessels of the liver, when thus deprived of their vaso-motory nerves, become distended, engorged with blood, hyperæmic; and the animals become glucosuric. Glucosuria thus induced continues for several days, and even for several weeks, as Schiff has shown by experiments on rats and rabbits; it is therefore different from irritative glucosuria, which is very transient. Paralytic glucosuria gives a fair picture of diabetes properly so called, that disease which is so rebellious under treatment. The diabetes produced experimentally by destroying the nervous centres, and likewise gangrenous diabetes, are paralytic forms of the disease.

Gentlemen, it is not only local lesions of the nervous system which promote the secretion of sugar in the liver: Claude Bernard has proved that this result is also caused by general nervous disturbance.

Similar results are also produced by excitement of the hepatic gland, occasioned through the medium of the digestive canal, or in a still more direct manner. Thus, Dr. Leconte, *Professeur Agrégé* of our Faculty, has produced artificial diabetes in dogs by poisoning them with the nitrate of uranium administered in small doses. Again, Dr. Harley, by injecting into the branches of the vena porta irritating substances, such as ether or a solution of ammonia, has observed that the injection, on arriving at the liver, produced there a local and direct excitement; and after some time, he has detected

[1] See p. 517.

sugar in the urine of the animal upon which he operated. Finally, I have mentioned to you, on the authority of Claude Bernard, a case of accidental diabetes, which supervened in an individual consecutively to a blow in the region of the liver; and I may add, that I have seen a similar occurrence in a man who received a kick in the right side from a horse.

But, an opposite effect is produced if the excitation of the gland is more than necessary to stimulate its secreting power, and amounts to irritation: the secretion of sugar is then greatly diminished. This is a fact in pathology to which I have more than once had occasion to call your attention.

It is to excitation proceeding unsuitably far that we must attribute the diminution of the saccharine secretion which takes place under the influence of fever, or of acute disease supervening in the course of saccharine diabetes. Under such circumstances, the glucosuria, as I have already mentioned, is sometimes temporarily suspended.

Finally, Gentlemen, excessive secretion of sugar in the liver is, as the older physicians would have said, the "proximate" cause of saccharine diabetes. It is unnecessary to seek for an explanation of this in the chemical reactions which take place in the digestive canal and blood-vessels, which are very different from reactions under our control, such as can be produced in experimental glasses; but to which, nevertheless, an attempt has been made to liken the reactions of vital chemistry.

Physiological facts find their explanation in the pathology of the disease which we are now studying. The influence of local lesions of the nervous system, or of direct excitement of the liver, explains the pathogenesis of diabetes when symptomatic of the cerebral or hepatic affections of which I have spoken. In those cases upon which morbid anatomy throws no light—and such cases are numerous—there is reason to believe that the diabetes is dependent upon disturbance of the nervous system reacting upon the hepatic secreting function. Though the nature of this disturbance may escape our observation, it at all events manifests itself by a variety of symptoms, such as gastric disorder, disturbance of the sensory, motory, and intellectual powers, as well as of the senses and organs of generation. These facts have a direct bearing upon the *treatment* of saccharine diabetes.

Once more, Gentlemen, let me impress upon you the importance of the fact, that diabetes must be considered both as a disease and as a

symptom, just as albuminuria may be either the consequence of that kind of *nephritis* which is called *albuminous,* or be an epiphenomenon common to many diseases in which there is no lesion of the kidneys. For example, both albuminuria and glucosuria may supervene in diseases of the liver, pancreas, lungs, brain, spinal marrow, or great sympathetic nerve.

Observation has shown that in these cases there may be an organic lesion of the liver, brain, or lungs, while in others, there is no appreciable material lesion; or that if any such lesion exist, it is only of a temporary nature, as is probably the case in epileptic fits and hysterical convulsions, as also, when patients are under the influence of inhalations of ether or chloroform.

In all these cases, we must suppose that there is a modification of the hepatic or pulmonary hematosis, and that this modification is sufficient to determine an intermittent or continuous passage of sugar into the urinary secretion. Cerebral lesions, particularly of the medulla oblongata, disturb the regularity of the respiratory functions; and likewise, when there is direct lesion of the lungs, there is imperfect hematosis. MM. Reynoso and Michéa have laid great stress upon this local etiology of glucosuria, both in relation to acute and chronic lesions. The same view may be taken of lesions of the liver, the other organ of hematosis, and the active agent in glucogenesis. It is easy to see that an organic lesion of the liver may so modify the function of the organ, as to cause sugar to pass into the urine in greater or less quantity according to the greater or less extent of the lesion, as is proved by the experiments of Claude Bernard and Schiff.

Glucosuria may also be produced by a modification of the circulatory apparatus of these organs, even when the modification is only temporary. Indeed, there is observed in fits of epilepsy and hysteria an asphyxial stage; that is to say, stasis of the blood in the lungs, in the right side of the heart, and probably also in the liver. This asphyxia would cause a diminution, or at least a modification of hematosis, which would account for the intermittent passage of glucose into the urine.

Moreover, there can be no doubt as to the part which the nervous system performs in the production of glucosuria. The physiological experiments upon the medulla oblongata, pneumogastric nerve, great sympathetic nerve, and spinal marrow, completely establish this fact:

and every day we find physiological experiment confirmed by clinical experience.

It is unnecessary to expatiate at greater length on this point. I have already gone into such details that I do not think that I need upon this occasion re-enter upon the subject.

To conclude, let me remind you that glucosuria not unfrequently exists in women who are pregnant or giving suck. For this interesting discovery, we are indebted to MM. Blot[1] and Reveil. In these cases, the sugar is never produced in great quantity: in nursing women, however, there has been found, on chemical analysis, as much as ten or twelve grammes in a thousand grammes of urine. These facts have been confirmed by other observers: but I ought to add, that M. Leconte[2] has arrived at conclusions different from those of MM. Blot and Reveil. It is, therefore, still an open question, and one to which, upon a future occasion, I must direct your attention.

I have now come to the question of *treatment*.

In the class of cases of which I have just been speaking, and in all those in which glucosuria is accidental and temporary, medical intervention is almost superfluous, because the affection will cease spontaneously, and, as a rule, in a short time. We have, therefore, only to consider in a special manner the treatment of persistent diabetes.

Diet is of the utmost importance. Clinical observation has shown that feculent food increases, while an almost exclusively animal diet diminishes, the quantity of sugar which diabetic patients pass in their urine. This is not because when we keep patients on an animal diet, we deprive them of the alimentary substances which furnish saccharine matter in greatest abundance. We have seen that saccharine matter is produced when the diet is animal as well as when it is vegetable, though in less quantity: we have also seen that sugar taken as aliment is transformed in the liver into a special substance very different from diabetic sugar; and that the latter is exclusively a hepatic secretion. An animal diet suits diabetic patients better

[1] Blot:—De la Glycosurie Physiologique des Femmes en Couches, des Nourices, et d'un certain nombre de Femmes Encientes. [*Gazette Médicale*; 1856, p. 720: and *Comptes Rendus de l'Académie des Sciences*; for 6th October, 1856.]

[2] Leconte:—*Comptes Rendus de la Société de Biologie*, for the year 1857, p. 60.

than a vegetable diet, because the latter, particularly when it is feculent, increases the excess of functional activity both of the liver and kidneys—it is because vegetable are much more diuretic than animal substances, as is shown by a much larger quantity of urine being passed by herbivorous than by carnivorous animals. The precept laid down by Rollo, and followed since his day, of giving diabetic patients a diet as nitrogenous as possible is in accordance with the teaching of physiology.

Nevertheless, Gentlemen, it is necessary to guard against adopting extreme views, and believing that diabetes demands an exclusively animal regimen, involving rigorous abstinence from every other kind of food. In fact, you will meet with diabetic patients who pass very little glucose in their urine, whilst they adhere to a regimen consisting only of green vegetables containing a large quantity of chlorophille, such as spinach, sorrel, cabbage, and cress; nay, even when they take acid fruits, such as currants, raspberries, and cherries.

In a disease in which disturbance of the nutritive functions plays unquestionably a leading part, it is of the utmost importance to avoid everything which might increase that disturbance; and consequently, it is necessary to vary the food so as not to induce loathing, a speedy effect of the exclusive use of the same kind of aliment.

Though a diet consisting chiefly of animal food is the most appropriate for diabetic patients, their regimen ought to contain a certain quantity of herbaceous vegetables, which are much more easily digested than feculent substances. I not only sanction, but I even recommend, the use of red fruits: failing them, I allow other fruits to be eaten, such as pears, apples, and even grapes, although they contain a large quantity of glucose.

Gentlemen, I cannot too emphatically raise my voice against the abuse of giving an exclusively animal diet in diabetes; and I must speak quite as strongly against the abuse of alcalies, which have been prescribed, particularly of late, as unquestionable specifics in glucosuria.

Though an exclusively animal diet immediately diminishes the thirst and excessive diuresis, it soon occasions intolerable loathing, and the health of the patients, which had seemed to be improving, is again deranged, and indeed becomes worse than it had been previously. On the other hand, however, if we rest satisfied with greatly diminishing the proportion of feculent nutriment, and allow

the use of fruits and green vegetables, both appetite and strength are maintained; and although there may be a large quantity of glucose in the urine, there is hardly any derangement of health. I have had diabetic patients under treatment for ten years who could not be supposed to be passing glucose, unless, from time to time, the urine were chemically examined.

I have no objection to patients eating a small quantity of bread. This is a point in respect of which I take largely into account the taste of the individual, not, for example, interdicting its use to that numerous class of persons who cannot eat unless they are allowed some bread. I recommend bread made of the flour of rye or wheat, and not bread made of gluten, which has a disagreeable taste, in reality has no advantage, and is only prescribed in accordance with a chemical theory.

Pharmaceutical means are useful in assisting us to obtain a better regulation of the digestive functions.

Alcalies are unquestionably beneficial. During last century, this fact was recognised: lime-water was the remedy then prescribed for appeasing the burning thirst, and diminishing the urinary secretion of diabetic patients. At the present time, the alcaline remedies in use are of infinite variety, whether we administer such medicines as the carbonate of lime, the bicarbonate of soda, or magnesia in the form of powder, or give natural mineral waters such as those of Vichy and Pougues, which contain alcaline ingredients in greater or less quantity.[1]

The unquestionable usefulness of these remedies in the treatment of saccharine diabetes must not be allowed to lead us to the conclusion that they act as alcalies, that is to say, by producing within the economy the same reactions which we see them produce in our laboratory experiments.

You are aware that sugars of the class *glucoses*, in which is comprised the sugar of diabetes, are destroyed by the caustic alcalies, potash, soda, lime, &c., being changed into peculiar brown acids, with a rapidity greater in proportion to the greater concentration of the alcali, and its greater elevation of temperature.

It is on this fact that some chemists have founded their theory of glucosuria and saccharine diabetes. They say that the absence of

[1] See the *Dictionnaire Général des Eaux Minérales et d' Hydrologie Médicale*: Vol. II, p. 563, 965. Paris: 1860.

sugar from the urine of a healthy man shows that the saccharine matter is derived from the aliment, and is destroyed by the blood which in its normal state is sufficiently alcaline to effect this transformation; and that therefore glucosuria is the result of the blood not being sufficiently alcaline to accomplish this change. I should not pause to recall to your recollection this chemical theory, victoriously assailed by Claude Bernard, had it not attained a widespread celebrity, had it not been received at first with a certain infatuation which is well remembered: for in fact, it has been refuted by the chemists themselves. First of all, it has been shown by Professor Poggiale, that to burn glucose in contact with alcalies a temperature of 95° C. is required; a fact which is of itself sufficient to overthrow the theory of which I have been speaking. In the second place, Professor Poggiale observed, that when glucose and an alcaline salt—the carbonate of soda or of potash for instance—were injected simultaneously into the vessels, the quantity of glucose which the animal experimented upon passed in its water was exactly the same as when glucose was alone injected.

Here then, the chemical explanation is at fault, as is always the case when we endeavour to apply chemical laws to the phenomena of the chemistry of the living body. Still, however, the clinical fact remains, that alcalies are unquestionably useful in the treatment of saccharine diabetes. Their action has a powerfully alterative effect upon the digestive canal, and imparts regularity to its functions: alcalies do not act by curing the diabetes, but by replacing the patients in certain conditions in respect of nutrition, in virtue of which the abnormally excessive production of sugar does not take place. This statement may seem almost a paradox. Let me explain what I mean. Under the influence of alcalies, the same takes place in respect of diabetes which takes place in respect of gravel. These remedies do not act by producing alcalisation of the urine, but by regulating the renal secretion.

Were we to accept the theory of alcalisation of the blood in diabetes, it would be necessary to administer alcalies in the greatest possible quantity, and indefinitely to continue to employ them. This, Gentlemen, would be an error most prejudicial to the patients.

I cannot too strongly impress upon you, that alcalies ought only to be given as adjuvants in the treatment, in moderate doses, and for not more than eight or ten consecutive days once a month.

Other medicines may be associated with them. Tonic remedies may be given, such as rhubarb, for example, during eight days once a month, in doses of from fifteen to twenty-five centigrammes after each meal.

In the wards of my lamented colleague Legroux, you saw a glucosuric patient who was simultaneously treated by arsenical preparations and hydropathy. Under this treatment, the state of the man to whom I refer became greatly ameliorated.

Hydropathy is a very powerful medication in the treatment of diabetes, by its action on the great organs of the economy; as is indeed every means by which the assimilative functions are stimulated.

I am unwilling to terminate this lecture, already so long, and so full of details which perhaps you have considered superfluous, without saying two words on the immense influence of *exercise*. A diabetic patient who takes daily very active exercise on foot, may, without in any way changing his regimen, temporarily regain his lost health. I have known glucosuric individuals, who, during the hunting season, have ceased to drink and to urinate in excess, have regained strength and appetite, and, in spite of their fatigues, have recovered virile power lost at the onset of the disease. Exercise cannot be too strongly recommended to these patients: it may be said, that when there is combined with a suitable, but by no means very severe regimen, the daily exercise of which I have just spoken, diabetes, particularly in fat persons, constitutes an indisposition rather than a very severe malady.

By means of a well devised hygienical system and regimen, aided by the judicious and prudent administration of medicines, we may hope to cure a few, and to relieve a great many, diabetic patients. I refer, of course, to patients who have not reached the last stage of the disease; for in that stage of wasting, the malady is beyond the resources of art.

LECTURE LXV.

POLYDIPSIA.

Cases.—Non-saccharine Diabetes may supervene in the Offspring of Polyuric, Glucosuric, and Albuminuric Parents.—Intercurrent Cerebral Affections may cause the Cessation of Glucosuria as well as of Albuminuria.

Gentlemen:—Some days ago, a man left the Hôtel-Dieu, who had been several months in our clinical wards for polydipsia. He presented, perhaps, one of the most remarkable examples which are to be met with of this affection. Although not completely cured, his improvement was so great, that, of his own accord, he asked to be allowed to leave the hospital.

The treatment to which this patient was subjected consisted in his taking the extract of valerian in quantities rapidly increased to very large doses. That mode of treatment afforded me still more satisfactory results in a previous case of a similar kind.

Perhaps some of you may remember having seen the patient to whom I refer in St. Agnes's ward. Like the man whose case is before us to-day, he was affected with polydipsia and polyuria. Every day, he drank thirty-two litres of tisane, and passed a proportionate quantity of urine. Professor Bouchardat, then principal apothecary at the Hôtel-Dieu, analysed the urine at different times, without finding in it the least trace of glucose. It is a remarkable circumstance that the skin of this patient's face was frequently the seat of very intense erythema, unaccompanied by fever, and coincident with excessive thirst and excessive urinary secretion: after two or three days, this erythema disappeared, but in a short time reappeared. In other respects, the man's health was good. I prescribed extract of valerian, which was by successive augmentations increased to the enormous daily quantity of 30 grammes [457½ grains]. There was, under this treatment, a simultaneous diminu-

tion in the thirst and in the urinary secretion; and after four months of the treatment, the cure was complete. When I saw this man at a later period, his health was very good.

In a similar case, reported by Dr. Rayer, the success of the treatment was still more rapid.

The patient, a young lad, was consumed by an unquenchable thirst: he urinated in proportion to the quantity of fluid he drank. His urine was almost as light as water, inodorous, colourless, insipid, and exceedingly profuse in quantity. The little patient did not grow thin, ate well, and, with the exception I have now stated, enjoyed perfect health. The polydipsia and the polyuria, both simple in their nature, were apparently produced by a nervous affection, essentially different from diabetes, and, in fact, having nothing in common with it, except the profusion of the urinary secretion. The symptoms yielded, within three weeks or a month, to valerian, administered in the form of powder, which, practically, is the same as giving the extract. Several methods of treatment, particularly the treatment by opium, had entirely failed. Dr. Rayer has employed the same treatment with advantage in other cases.

Although the result has not quite realised my expectations in the case which has supplied the subject of the present lecture, it does not the less deserve to be taken into consideration by you.

In our patient, the disease had begun, according to his own account, four years previously: it was recognised under the following circumstances. The young man, then twenty, had been admitted to the surgical wards of my honourable friend and brother, Dr. Langier, for a symptom which was in itself insignificant. He was in the habit of sometimes complaining of pains in the lumbar region, particularly in the right side, pains having no relation to the surgical affection for which he came into hospital, and of which he was able to give a very imperfect account. It was evident, however, that he drank a great deal, and urinated proportionately. He was then drinking about six litres a day, and, according to his own expression, making as much water as any four men. This circumstance attracted the attention of M. Langier, who begged M. Bouchardat to examine the urine. It was found to contain sugar, but in small quantity. After some time, the patient was transferred to the clinical wards of Dr. Rostart, where he only remained five weeks. When he left the hospital, the quantity of drink which he took in the twenty-four

hours varied between 18 and 20 litres, and the urine he passed amounted to 25 litres.

He was soon obliged to seek admission at La Charité. He was then passing daily as much as 32 litres of urine. In the wards in which he was placed, he was subjected to an almost total abstinence from drinks; and to quench his craving thirst, he was given some ice or lemon to suck, allowing him to take food in accordance with the promptings of his appetite. The unfortunate young man resigned himself to this severe regimen, which he endured for eight months, although he suffered so cruelly from it that one day he seized the chamber-pot, and drank the contents to the last drop! It may be said with truth, that under the influence of the regimen his state was greatly ameliorated, in this sense, that the urine he passed did not exceed 10 pounds in the course of the twenty-four hours. Nevertheless, as he felt his strength decreasing, his sight failing, and his body wasting, he asked to be allowed to leave the hospital.

He remained at home for a year; but at the end of that period, the pain in the kidneys having returned, the thirst having become very intense, and the quantity of urine very great, he was admitted to the Lariboisière Hospital. At that time, he was daily drinking 14 litres of fluid, and passing from 18 to 20 litres of urine. Some traces of sugar were detected in the urine. A plan of treatment was instituted, the basis of which consisted in administering preparations of iron, opium, and cinchona; and giving a diet consisting chiefly of animal food, from which feculent substances were excluded, and in which bread made of gluten was substituted for common bread. This treatment did not produce the slightest influence on the disease: it did not diminish the quantity of sugar (which was small) in the urine; and the amount of urine passed was greater than before. At the end of ten weeks, the patient left the hospital, in no degree benefited by the treatment; but in a few days, he was readmitted, when he went into the wards of my excellent friend Dr. Pidoux.

Dr. Pidoux prescribed for him a diet consisting of five pounds of animal food, of which three pounds were ham, and two pounds roast meat: he was also ordered toasted bread, and a litre of wine in addition to the ordinary rations of convalescent patients. He was likewise put on bicarbonate of soda, and powder of valerian, of which latter he took 10 grammes daily from the first day.

There was soon a diminution in the quantity of urine: it fell from 29 to 11 or 12 litres in the twenty-four hours.

The extraordinary ease with which this man bore alcoholic drinks independently of his wine—he took 6 litres of a vinous tisane—induced Dr. Pidoux to try the effects of brandy. Within two hours, and at intervals of half an hour, he took a litre of what is known in commerce under the name of "*trois-six*:" he every day took the same quantity without appearing to be in the least degree inconvenienced by so doing. He stated that from the beginning he had acquired so great an immunity as to be able to drink large quantities of stimulants, without feeling the slightest symptoms of being drunk. On several occasions, he took for a wager twenty litres of wine, gaining his wager without producing any effect on the nervous system.

After remaining three months in Dr. Pidoux's wards, he felt able to resume his employment, one involving hard work and fatigue, as servant to a horse-dealer.

But once more, the amendment was of short duration: some months later, he returned to our wards.

I resumed the valerian treatment. I began at once by giving 10 grammes of the extract in the twenty-four hours; and I progressively increased the quantity till I reached 30 grammes. The result was a speedy diminution in the quantity of urine from 29 to 6 litres, the thirst proportionately abating. Unfortunately at last, the tolerance for the valerian was lost, and as soon as taken, it was vomited. The patient began to lose his appetite, and in place of eating four times as much as an ordinary man, which he had been doing, he was satisfied with "the four portions" of the hospital, which represent about 50 décagrammes [1 lb. 1 oz. and 10 drachms] of bread, 20 décagrammes [6 oz. and 10 drachms] of meat, and 50 centilitres [1 pint and 12 fld. oz.] of vegetables. Some convulsive phenomena having showed themselves, it became necessary to suspend the treatment. At the end of a fortnight, he was passing 16 litres of urine in the 24 hours.

At this period, the patient, by permission, spent some days at home: on the fourth day, he returned to the hospital, drinking 33 litres in the 24 hours, and passing from 37 to 43 litres in the same space of time. Not daring to resume the valerian, I tried belladonna in doses of one centigramme; and—strange to tell!—this man, who could drink 20 litres and a litre of alcohol of sp. gr. 0·835 [90 *degrés*

centésimaux][1] without being intoxicated, experienced violent effects from this minute dose of belladonna; and each time it was repeated, similar results were produced. I then had recourse to preparations of strychnia, medicines which render such great services in nervous diseases. The syrup of the sulphate of strychnia not being tolerated, I gave the tincture of nux vomica, which also I was obliged to discontinue, although in the first instance, under the influence of this medicine, the quantity of urine fell from 37 to 18 litres.

Then, after allowing the patient to rest for a time, I resumed the valerian, which I again caused to be administered to the extent of 10 grammes a day, as on the first occasion of my prescribing it; but I did not increase the dose to more than 12 grammes. In twenty-five days, there was a manifest improvement. The thirst had considerably diminished, and there was much less urine passed. When the man left our wards, he had for some time not been drinking more than four, three, and latterly, two and a half litres a day, while his daily quantity of urine had gone down to five, four, and at last even to three litres and a half. This patient was completely impotent, like the generality of polydipsic and diabetic patients.

The history which I have now related presents some resemblance to the case of the patient of whom I spoke in my last lecture on saccharine diabetes, that patient at whose autopsy Dr. Luys found a lesion of the fourth ventricle. In neither of these patients was the polyuria entirely simple, for both had glucosuria at the beginning of the malady.

Now, there is no glucosuria in polydipsia properly so called, in that disease known by the names, *false diabetes, insipid diabetes, hydromania, polyuria,* and *urinæ profluxio.*

In polydipsia, the urine is clear like water, and never contains a trace of sugar: its density, in place of being greater than that of normal urine, in place of rising to 1,030 or even to 1,074, goes down to 1,009 and 1,001.

The quantity of urine passed in the twenty-four hours is always much greater in polydipsia than in glucosuria; and, though in excess of the quantity of fluid taken by the individuals, it nevertheless bears a relation to it. Thus, the patient in St. Agnes's ward, who

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[1] "*Un litre d'alcool à* 90 *degrés centésimaux:*" rather more than a quart of alcohol at sp. gr. 0·835, or 57½ over proof, which is a trifle stronger than the rectified spirit of the British Pharmacopœia.—Translator.

between two of my visits was drinking six, eight, fifteen, and up to forty litres of fluid, passed within the same period, eight, ten, sixteen, thirty-seven, and even forty-three litres of urine in the twenty-four hours.

Another polydipsic young man, who died in our wards with purpura, had had sugar in his urine at the beginning of the malady, like the other patient of whom I have just spoken. There is, therefore, a relationship, which cannot be ignored, between glucosuria and polydipsia, a fact which is in harmony with the physiological experiments of Claude Bernard. The illustrious Professor of the *Collége de France*, by irritating certain parts of the floor of the fourth ventricle, produced sometimes albuminuria, sometimes saccharine diabetes, and at other times polyuria.[1] Is it not, therefore, very probable, that a perturbation of the nervous system, the essential nature of which is still unknown, is the principal cause of these three maladies which at a first glance seem so entirely distinct from one another?

As I have just told you, polyuria, saccharine diabetes, and also sometimes albuminuria, may, in succession, attack the same individual: it is also not unusual to see suffering from non-saccharine diabetes, children whose predecessors were either glucosuric or albuminuric.

I was lately attending, in consultation with Dr. Bergerd, my excellent friend and colleague in the hospitals, a polyuric young lady, whose case I shall now relate to you in a summary manner.

When I saw her, she was nineteen years of age, and had all the appearance of perfectly good health. She was the grand-daughter of a diabetic subject, who had had glucosuria for ten years, without any notable derangement of health. She had great obesity. I have already told you, when speaking of saccharine diabetes, that it is a more common disease among fat than thin persons, and that to compensate, as it were, for this peculiarity, it exercises a much less disastrous effect upon the constitution of the former. The glucosuria was rapidly diminished at the onset of the disease by the use of alcalies; but then it reappeared. It lasted ten years, as I have told you, and ceased suddenly and definitively upon the day on which he was seized with cerebral symptoms, due probably to cerebral

[1] Claude Bernard:—Leçons de Physiologie Expérimentale Appliquée à la Médecine.

hæmorrhage followed by softening of the brain. In the eighteen months, during which the symptoms continued, the patient became exceedingly thin.

Gentlemen, let me pause for an instant to call your attention to this fact—this strange coincidence. Does it astonish you to see lesions of the encephalon sometimes producing saccharine diabetes, as in the cases I quoted when lecturing on glucosuria, and in the experiments made on animals by wounding the fourth ventricle, while in other cases, the malady ceases, upon the production of cerebral lesions of another kind?

I shall afterwards have to call your attention to facts of this description in relation to albuminuria. You ought to remember a man, aged 57, who lay in bed 14 of St. Agnes's ward. He had Bright's disease, with general anasarca, and pulmonary infiltration. I did not believe that he could live a month. He was suddenly struck by hemiplegia: the albuminuria disappeared: the general health was restored. Some months later, when I sent the poor paralytic to the Bicêtre, there had been no return of the albuminuria since the occurrence of the cerebral hæmorrhage. It is a remarkable fact, that during my long and busy medical career, I have only seen three recoveries from confirmed Bright's disease; and in all the three, the albuminuria ceased, and the general health became re-established. This took place in one of the cases upon the patient becoming a decided epileptic: in the two other cases, recovery followed attacks of cerebral hæmorrhage which had left the patients hemiplegic. This is an additional proof of the immense influence of the nervous element in the production of albuminuria, saccharine diabetes, and polydipsia.

Let us now return to the case of the young girl. Her grandfather, as I have said, was diabetic. One of her uncles died of Bright's disease. She herself had, in childhood, always been delicate, or *lymphatic*, to use the common expression. In May, 1856, when she was fourteen years of age, she showed symptoms of chlorosis: soon afterwards, ardent thirst supervened: the urine became aqueous, its specific gravity being hardly higher than that of distilled water: the general health was radically deteriorated. Under the influence of valerian and chalybeates, her state improved, her strength returned, and the quantity of urine passed in [the twenty-four hours fell from ten to six or seven litres. Soon, the urine was down in quantity to three litres and a half; and up in specific gravity from 1·003 to

1019. From the year 1856 to the year 1862, the polydipsia continued, increasing and diminishing, however, without its being always easy to assign a reason for the change. Mineral waters, sea-bathing, hydropathy, and valerian afforded temporary amelioration. The patient has become well developed, tall, and plump: but the catamenia have not appeared, although different kinds of emenagogue treatment have been perseveringly employed.

Gentlemen, the only morbid phenomena of the first stage of polydipsia are excessive, sometimes inextinguishable, thirst, and the emission of an abnormally large quantity of urine. However—notwithstanding assertions to the contrary by the majority of authors, who on this point have only repeated the statement made by the first—along with the thirst, the appetite is not only usually augmented, but becomes exceedingly increased. You recollect the frightful quantity of aliment consumed in the twenty-four hours by our patient of St. Agnes's ward: you have heard of the terror he inspired in the keepers of those eating-houses where bread is allowed without extra charge to the extent of each customer's wishes. I have been told, that after he had taken one or two meals at one of these eating-houses, he was presented with money to prevent him coming back to dine.

With the exception of this ferocious appetite, and the burning thirst, the digestive functions do not seem to be at all disturbed. Digestion is accomplished with perfect regularity: the general health continues good. In such cases, polydipsia constitutes a very inconvenient infirmity rather than a disease.

In some cases, it is a fleeting symptom, but in others it is a malady which lasts as long as life. It sometimes makes its appearance in childhood, becomes more strongly developed at puberty, and then continues, resisting all curative measures; or, should treatment moderate the symptoms, or cause the affection to cease for a time, a complete cure is almost never obtained.

But do not suppose, Gentlemen, that matters remain long in so favourable a position. Insurmountable anorexia, diarrhœa, and wasting soon succeed the boulimia, the symptoms becoming more and more alarming: the skin acquires a withered clay-like appearance, the breath becomes fœtid, and symptoms of tubercular phthisis show themselves just as in saccharine diabetes.

For a long time, Gentlemen, adopting the opinion of my predecessors, I believed that polydipsia was a less serious disease than

glucosuria; but that is a point in respect of which experience has greatly modified my views. Having seen in my private practice and hospital wards a great number of glucosuric patients retain good health for a long time, although I did not employ any very active treatment, I have, on the other hand, had the pain to see nearly all the polyuric patients whom I had to treat, waste away rapidly, and die much earlier than those who had saccharine diabetes. In the majority of glucosuric persons, let me add, that I can easily modify the quantity and character of the urinary secretion, while I find that I can be of little use to polydipsic patients. The young lady whom I saw with my honourable friend Dr. J. Bergeron (and of whom I have just been speaking), is a fresh proof that there are some fortunate and rare cases, in which polydipsia does not greatly disturb the general health, even while it resists, with disheartening obstinacy, treatment the most diversified and the most rational.

If sometimes this remarkable affection has as an evident starting-point, strong mental emotions—if it be a not unusual epiphenomenon of certain nervous affections, particularly of hysteria—still, generally speaking, its causes are utterly unknown.

The antispasmodic, or to use a more appropriate expression, the *valerian* treatment, is the best treatment for polydipsia. At least such is the conclusion to be drawn from the case of the patient who has just left the clinical wards—from a similar case which I treated in a similar manner six years ago—and from the facts reported by Dr. Rayer. Hydropathy has also seemed to have been of great use in some cases.

LECTURE LXVII.[1]

VERTIGO A STOMACHO LÆSO.[2]

"Vertigo ab aure læsa."—"Vertige Labrinthique."—*Stomachic Vertigo is often mistaken.—Symptoms which characterise it often considered to depend on Cerebral Congestion, and consequently the Treatment adopted often aggravates it.—Vertigo depending on Lesions of the Labyrinth resemble Stomachic Vertigo.—Treatment of Stomachic Vertigo is that of Dyspepsia.*

Gentlemen:—A woman aged fifty-seven, having symptoms to which I wish to direct your particular attention, has occupied bed 29^{bis} in St. Bernard's ward for about two months.

This patient generally enjoyed good health, till she reached the critical period of life, when she suffered much from menorrhagia, by which her strength was greatly enfeebled. At the same period, she likewise began to suffer from a feeling of weight and pain in the epigastric region, and from pain shooting through the abdomen, back, loins, and precordial region, in which latter situation, she felt tingling sensations. She retained her appetite, but had a disgust for farinaceous vegetables, and cold food, which increased the feeling of weight in the epigastrium. The digestion of cold food was difficult, and attended by general discomfort and flushing of the face. Warm food, on the contrary, calmed the pains which arose during the intervals between repasts.

Gentlemen, let me repeat the history given by the patient of the symptoms to which I now wish to call your special attention.

Four days before her arrival at the hospital, she had left her native place, where for six previous days, she had been subjected to much

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[1] Lecture LXVI, will be found in the First Volume of the Translation of the New Sydenham Society. See "Notice to the Reader" prefixed to the Second Volume.—Translator.

[2] Vertigo Stomacal.—Vertigo per consensum ventriculi of the old authors.

fatigue and mental worry. Without feeling at that time exactly that she was an invalid, she observed, that she was losing her appetite, and that her digestion was becoming unusually painful.

On the day of her leaving, after having made her usual breakfast on coffee and milk, she set out to walk the three leagues which separate her village from the town of Saint-Quentin. She had formerly, more than once, accomplished this journey without being at all fatigued; and this was even the case since her arrival at Paris six days previously. She was proceeding on her way, in company with one of her children, when, after having walked hardly three kilometers, she was all at once seized with giddiness and weight in the head, unaccompanied by severe pain, eye-dazzling, or any syncopie tendency. It seemed to her, that the earth was opening in front of her, her legs bent, and she felt as if being irresistibly borne towards an open abyss which she believed she saw at her feet. This singular hallucination was accompanied by nausea, and a desire to vomit: in fact, she did bring up some of the food which she had taken in the morning, and subsequently, a small quantity of clear fluid. She nevertheless retained unimpaired her consciousness, and was perfectly aware of the error of her senses, at the very time she was screaming with terror and beseeching her son to prevent her from falling into the abyss. This extraordinary condition lasted ten minutes. In describing it to me, she said that her sensations might be compared to those experienced when looking down on the ground from the top of a steeple. Her giddiness was so violent, that to prevent her from falling, it was necessary to make her sit down, and afterwards lie down on a bed brought to her from the nearest house. The sensation of weight in the head was soon succeeded by acute pain: to use her own expression, "she felt as if her skull was being split." For several hours, she was unable to bear the movement of a carriage, so that it was necessary to leave her for some hours in a house where she had been hospitably received. During the evening, the symptoms seemed quite to disappear, and the patient felt well enough to return home on foot: in fact, she was the better of the walk, for on reaching her residence, the only remaining symptom of her attack was a little heaviness of the head: she ate well, and had nine hours of very tranquil sleep.

On awaking next morning, she complained of a little heaviness of the head and feelings of numbness; but she breakfasted as usual, and then again started for Saint-Quentin. After accomplishing this

walk of three leagues, feeling quite re-established, she left by rail. She had scarcely gone half the journey, when she was seized with symptoms precisely similar to those of the previous evening. At the Creil station she got out of the carriage, when the giddiness was so great, that to avoid falling, she had to lean on the arm of a fellow traveller. The carriages, she told me, seemed as if they were dancing, as if they were being carried high up into the air and then let fall, to be engulphed in the earth which seemed to open; and she felt herself as if being drawn into the abyss. She continued to experience these sensations for nearly ten minutes; and for a long time afterwards—even at the time she described them to me—she retained the impression of terror which they had originally caused. As on the previous evening, however, she was perfectly aware that the whole was a delusion, and gave clear replies to the questions addressed to her. She returned to the railway carriage, retaining, however, some degree of general discomfort, and from a fear of increasing this uncomfortable state, she remained silent during the remainder of the journey. On her arrival at Paris, two hours afterwards, the giddiness did not return, but the state of general discomfort continued, nor had it left her, when she came next day to the Hôtel-Dieu to visit her daughter, who was a patient there. She had then also pain in the head, which she described as resembling a sensation of having her forehead split. In accordance with the recommendation of her daughter, she asked to be admitted as a patient, and was received into our wards.

On examining the abdominal region, I found that there was slight enlargement of the liver; but there was no trace of jaundice, ascites, or anasarca. On making pressure over the pit of the stomach, there was produced an increase of the pain, which the patient said never left her.

Rest, restorative diet, and a methodical use of very simple measures soon caused the symptoms to disappear. To-day, the woman is so thoroughly recovered, that she wishes to return to her home.

What had this patient? You remember, Gentlemen, what my diagnosis was from the first. I told you that the singular phenomena which she described to us depended upon the stomach; and that we had to do with that kind of vertigo which I have designated *vertigo a stomacho læso*, the same affection which the old authors denominated *vertigo per consensum ventriculi*, names for which my pupil, Dr. Blondeau, in a memoir on this subject, published at my

suggestion, has substituted "*vertigo stomacal*," a term which is less regular but much shorter.[1]

This is perhaps the most common kind of vertigo: it is at least that for which we are most commonly consulted in private practice. The obstinacy of the symptoms which characterise it, its constantly returning, and its seeming seriousness strangely torment those affected with it, deceive the persons about the patients, and often mislead even the medical attendants, who, misconceiving its nature, resort to treatment diametrically opposed to that which is appropriate. Giddiness originating in the stomach is often imputed to supposed cerebral congestion, a mistake leading to antiphlogistic treatment by bleeding, leeching, purging, and rigorously low diet—means which, in place of curing the disease, increase its severity; whereas, speedy benefit is obtained by suitable restorative diet and tonics.

The vertiginous phenomena, however variable they may be in their forms, have a something about them which is peculiar; and an experienced, attentive observer will suspect the nature of the symptoms, even when they do not present anything essentially characteristic of the affection of the digestive functions with which they are associated.

The phenomena to which I refer are sensations of emptiness and swimming in the head; or sometimes, the patient feels as if his temples were being tightly clasped by an iron ring. Presently, he experiences a sensation of icy coldness. Some patients tell us that they have a mist before their eyes, and that the objects which they see present a confused diversity of colours: others describe a large black wheel as moving before them with excessive rapidity. But the form of this affection which you will most frequently meet with is that to which the name *gyrosa* has been applied; when the individual is standing, everything about him seems to be whirling round: he is obliged to shut his eyes, and remain absolutely motionless, for he feels his legs tottering and bending under him, as if he were going to fall; and sometimes, indeed, he does fall. If he be lying down, he thinks he sees his bed revolving on an axis passing through his head and feet; or it may be, that the patient sees himself involved in the rotatory movement.

It is a remarkable and essentially characteristic fact, that to what-

[1] Blondeau (Léon):—*Archives Générales de Médecine* for September, 1858.

ever degree these symptoms are present, the patient never loses consciousness of his actions: even when he falls, there is no loss of consciousness, and he never misunderstands the nature of the odd sensations and hallucinations which terrify him. You recollect what we were so often told by our patient of St. Bernard's ward: she said that though it was impossible for her to shake off the feeling of terror at seeing the open abyss at her feet, although the terror was renewed even by the recollection of this sight, she knew perfectly well that it was an illusion of the senses. Other persons have told me of their having had analogous hallucinations, they knowing them at the time to be mere illusions.

The vertiginous phenomena are generally accompanied by squeamishness, which the sufferers compare to sea-sickness. It is indeed *nausea* in the literal acceptation of the word, which is derived from *ναῦς*, the Greek for a ship.

The slightest cause may bring on these vertiginous affections. The immediate cause of their development may be a trellised wall, a range of bars, or a striped pattern in the hangings of a room. The trellis, the bars, or the stripes look commingled, as if in a sort of mist, and they have a dim appearance. A rather sudden movement, or raising the head, may suffice to induce the phenomena. It is an interesting peculiarity, worthy of being noted, that, in general, the phenomena do not occur when the head is *lowered*, which is the reverse of what occurs when the vertigo depends on congestion of the encephalon.

These phenomena being constantly reproduced by the slightest cause, or even without any immediately exciting cause, get so strong a hold of the minds of the patients, that they, forgetting as it were their other dyspeptic symptoms, only think of, and only mention to the medical man, those which we are now considering. You have observed that the woman in St. Bernard's ward, and two patients in St. Agnes's ward (whose cases I shall have to refer to immediately), spoke only of the vertiginous symptoms, and made absolutely no mention whatever of the signs of disorder in the functions of digestion, to which, nevertheless, they were referable.

The gastric disorder, however, is usually indicated by well-marked symptoms. There are pains in the stomach, which are most violent after the ingestion of food, particularly of some kinds of food: they are increased and propagated to the back by pressure with the hand over the pit of the stomach: there is a feeling of weight, of cramp,

of acute pain shooting through the chest and abdomen, extending even to different parts of the body, and accompanied by a sensation of heat or burning in the region of the stomach. There are flatulent symptoms, acid eructations (not inodorous in general), and vomiting of a glairy mucous character, or sometimes of food. Constipation is more common than diarrhœa, although both may alternate in the same individual.

These symptoms are absent in a few exceptional cases; but if you interrogate the patients on the subject, you will almost always hear them complain of their digestion being slow and laborious.

But bear in mind, that, generally speaking, the vertiginous symptoms do not occur during, but long after digestion, that is to say, using the vulgar expression, when the stomach is empty. Here let me mention a fact bearing upon the treatment of this singular affection: the administration of a small quantity of food, such for example as a cup of broth, or a biscuit soaked in a little wine, will often prevent he symptoms from supervening, and stop them, should they have set in.

It would, nevertheless, appear, that under certain circumstances, eating may be the determining cause of the symptoms, if we may udge from what we saw in the two patients in St. Agnes's ward, to whom I have just referred.

One of these patients was a young man of twenty-five years of age. He stated that he had generally enjoyed good health, having never had any ailment except an affection, which was probably syphilitic, as he had been treated for it in the hospital for venereal cases: beyond this fact, he gave no precise information regarding that illness.

The following is the account he gave of the symptoms for which he came into the hospital. For a month, he had suffered from headache and pain in the right side: these were the symptoms which had chiefly annoyed him. He had abdominal pain, which was increased on pressure; but on making a very careful examination, I was unable to discover any sign of visceral lesion. The peculiarity in the abdominal pain was its subsiding spontaneously, immediately after a repast: but the patient had also cerebral symptoms which made him very anxious. They consisted in dizziness, impaired vision, buzzing in the ears, heaviness of the head, and a peculiar feeling which he compared to incipient drunkenness. He affirmed that he was not in the habit of drinking either wine or any other intoxi-

cating liquors to excess. When he attempted to rise from table, his legs bent under him, he experienced a feeling of general discomfort, and felt as if he were going to faint: to save himself from falling he was obliged to lean on whatever was within his reach. He never, however, lost consciousness.

These symptoms, which recurred, I repeat, after the patient had eaten, which recurred several times within the first hour immediately following a meal, were unaccompanied by pain or uneasy sensation in the region of the stomach, and constituted with the gastralgia, or rather enteralgia of which I have spoken, the only symptoms characteristic of the disorder of which the digestive functions were the seat.

It sometimes happens, though not often, that the vertiginous symptoms are not only the predominating morbid phenomena, but are the only ones of which the patients complain, and that because they suffer from none other: the dyspepsia in which they originate does not otherwise show itself. Here is a case in point.

A lady of a certain age came to Paris from Bordeaux to seek medical advice in respect of cerebral symptoms with which she had been distressed for several months, and which consisted in giddiness which scarcely allowed her a moment's tranquillity. This giddiness was brought on by the slightest cause, and proceeded so far as to produce a syncopic state, obliging the patient to keep the recumbent position. The bustle of the streets, the sight of the passers-by, or of a carriage going along at a rather quick pace, so decidedly caused the symptoms to return, that this lady was soon unable to leave her own room. She believed that she was threatened by an attack of apoplexy, and her fears were increased by those around her: their officious advice fanned her morbid fancies. To avoid the congestion which she dreaded, acting in harmony with her own theory that nothing should be done to augment the quantity of blood, she condemned herself to a very severe regimen, and lived exclusively on soups and beef tea. Her appetite decreased; but digestion continued to be properly performed. By pursuing this regimen, she was reduced to a deplorable state of cachexia. When my friend Dr. Lasègue was called in, he was struck by her emaciated appearance and yellow skin. At first, he thought that there was cancer, but minute examination disclosed no sign of any such affection: he soon became convinced, that the vertiginous symptoms depended on derangement of the functions of nutrition, and were increased by absti-

nence. Dr. Lasègue asked me to see the patient with him in consultation: I did so; and entirely concurred with his diagnosis. The result showed that we were right. By the administration of tonics and a restorative diet, in eight days, a remarkable change in her state was effected. The attacks of giddiness were much less frequent; and in a short time, the patient ceased to be troubled with them. In six weeks, she was well and plump.

You perceive, therefore, Gentlemen, that vertigo often supervenes in persons whose digestion is not at fault in any way. The appetite is good, the alvine evacuations regular, and there are no acid eructations; but nevertheless, success attends the employment of treatment directed against dyspepsia. I have frequently asked myself, whether the treatment which in these cases I directed against the affection of the stomach was not, unknown to me, addressed to the nervous system; and whether I had not diagnosed a gastric affection, rather from the effect of treatment, than from the symptoms of the disease; whether I had not been led into an error in diagnosis, by obtaining success from treatment usually employed with benefit in dyspepsia.

I know very well, that nausea and vomiting often occur in cases of vertigo, when the tissue of the stomach is in a perfectly normal state, and when its secretions are exactly what they ought to be; but who is not aware that in numerous affections of the nervous system, vomiting is a common symptom: without speaking of cerebral fever, let me mention sea-sickness, and the dizziness produced by waltzing, in both of which the gastric symptom is that which chiefly occupies the patient, and of which he complains the most bitterly. Now, in the vertigo which is called, rightly or wrongly, stomachic [*stomacal*], the nervous symptoms, and those observed in connection with the stomach, do not, for example, greatly differ from those of vertigo depending on lesions of the labyrinth.

It is right to state, however, that the latter form of vertigo very seldom yields to the treatment which is generally successful in simple vertigo, accompanied only by nausea and vomiting.

Stomachic vertigo occurs pretty frequently during convalescence from long illnesses, such as serious fevers, particularly when they have caused a great disturbance of the functions of nutrition, reacting on the digestive canal, while it still remains in a flagging state.

This occurred in our second patient in St. Agnes's ward.

This man was forty-eight years of age. He was of a robust con-

stitution, and had always been in good health till within fifteen months of his coming into our hands, when he was seized with severe scurvy caused by the miserable diet to which poverty condemned him. He lived in a very unwholesome lodging: his only room was badly ventilated, and being immediately under the roof, was exposed to cold, damp, and all vicissitudes of weather. I had an opportunity of seeing this man in another service in this hospital to which he had been admitted. There were large ecchymotic spots over the whole surface of the body, some of which were as large as the hand: the gums had been destroyed by sanious ulcerations: and there was great diminution of strength. The disease continued two months; or at least at the end of that period, the patient left the Hôtel-Dieu. For more than three months after this, the patient had obstinate lienteric diarrhœa: his appetite, however, remained good, and he ate a large quantity of food, but it was not aliment fitted to restore the waste of the body, being chiefly bread, and soups made without any meat. Digestion was performed badly: two hours after a meal, the alimentary substances were passed by stool almost in the state in which they had been swallowed: the stools were as many as twenty during the twenty-four hours. For five months, the general debility was so great, that the man could not leave his room. From the extreme degree to which emaciation had proceeded, he was unable to sit, and had therefore to remain in bed.

As soon as he was able to leave the house, this unfortunate person resumed his ill remunerated labour, that he might obtain sustenance for himself and family. Under these circumstances, his food was very inadequate: digestion, too, was imperfectly performed, in consequence of his having lost a great number of his teeth, and those which remained being so loose in their sockets from the scorbutic affection, that he could not masticate his food. His new lodging, though less unwholesome than the former, was still a very insalubrious abode.

Hygienic conditions of so unfavorable a description were not calculated to restore rapidly his lost strength. When he was readmitted to the hospital, he was still complaining of being very weak; but the cause of his coming back to the Hôtel-Dieu was a return of the symptoms from which he had suffered, and which occasioned him great anxiety in consequence of their having become much aggravated during eight preceding days.

I shall now tell you what he complained of. He felt numbness,

a sort of paralysis of the muscles which move the jaws, and of the tongue: this interfered with the opening of the mouth, and prevented him from being able to articulate certain words. His voice was hoarse, and he could no longer cry his wares on the streets as a costermonger. When I asked him to open his mouth, he was only able to expand his jaws half way; and that occasioned pain in the temporo-maxillary articulations. His tongue, which he could not protrude beyond the dental arch, obviously deviated to the right. On examining the back part of the mouth, I discovered that there was deformity of the veil of the palate which was not regularly concave: on the left, it was flattened, and the uvula was warped on that side; however, on touching the parts with a bristle, or the nib of a pen, it was seen that motor power remained, and that muscular contractions were excited.

For eight days, the patient had suffered from headache, which was uninfluenced by the stomach being full or empty: he compared the headache to that experienced after a drunken debauch. In relation to drinking, he affirmed, that he never took brandy, and very seldom drank alcoholic liquors of any kind. He added, that his brain was disturbed by less than one tumbler of wine, of which, in former times, he could easily have taken three litres without feeling any discomfort.

He slept well, and on awaking his head was free, but after sitting up for some minutes, the pain returned. He felt better when lying down, provided the head were placed rather high. When standing, he experienced tingling sensations in the eyes, and then disturbed vision: he saw ascending and descending sparks, and objects seemed to be turning and dancing around him: ere long, the sight became exceedingly dim, and he felt as if in a mist. To prevent himself from falling, he was obliged to lean on any support which might be nearest. Upon one occasion, he did fall; but there was no loss of consciousness, and, in a few seconds, he rose without assistance. These vertiginous symptoms increased when he raised or lowered his head: but they were less severe when he looked up than when he looked down, which is the reverse of what is usual in such cases. For some time, he tried to serve masons, but was obliged to renounce the attempt, in consequence of inability to mount scaffolding, without feeling as if his head were going round, a symptom to which he had not been subject before his illness.

When the vertigo supervened, the headache increased, and the patient felt as if his head were splitting: sometimes he had nausea,

but he never vomited. He stated, that neither before nor after eating had he ever had what could properly be called pain in the stomach.

The lienteric diarrhœa, by which he had been tormented for more than three months, was succeeded by constipation to such a degree, that he was eight days without a stool. For some time, however, the movements of the bowels had resumed their usual regularity.

In addition to the general debility of which he complained, he had a constant feeling of weight in the lower extremities: neither exercise nor the warmth of bed could warm him. Before he had the scurvy, he perspired easily in the feet; but now his feet remained dry, when all the other parts of the body were covered with sweat.

Rest for a week, good food, and medical treatment, consisting chiefly in the administration of alcalies, sufficed to stop the vertiginous symptoms; and this man felt so well after being ten days in our clinical wards, that he then expressed a wish to return home to his family.

Whatever relations this case may bear to the subject now before us, it is one of considerable complexity, for the vertigo may be considered as the result of profound derangement of the nervous system during a long and serious illness. According to this hypothesis, the symptoms were of the same kind as the paralysis of the veil of the palate, and the paralysis of the tongue, observed, as I have had occasion more than once already to tell you, as a sequel to certain diseases, such as diphtheria and typhoid fever, which produce a great strain upon the system. In this case, however, it was evident that the digestive organs were the organs more particularly involved, as was shown by the long continuance of the lienteric diarrhœa. So dependent were the vertiginous symptoms upon disordered digestion and nutrition, that good alimentation for a week was sufficient to effect a cure.

Nevertheless, Gentlemen, this case has not that distinctiveness which characterises those we most frequently meet with in practice. Some time ago, I was called to attend upon a magistrate, sixty years of age. Consequent upon very assiduous mental work, generally performed after dinner, he had been feeling a weight at the stomach, and frequent acid eructations. The appetite was becoming less and less every day. All at once, when looking up to the ceiling, he became exceedingly giddy, saw objects whirling round him, and felt

at the same time transient nausea. Being anxious about himself, he sent for his medical attendant, who prescribed purgatives and sinapised foot-baths. The malady, however, made rapid progress. There was vertigo not only when the patient was sitting or standing, but also when he was lying in bed. He had incessant nausea, which he compared to sea-sickness.

He was in a state of extreme anxiety, and believed he was threatened with apoplexy. Some physicians thought that there was incipient softening of the brain.

My opinion was that it was a case of stomachic vertigo. The treatment which I instituted consisted in administering alcalies and bitters: in a fortnight, the symptoms had entirely ceased. Some weeks later, they returned; but on a repetition of the treatment, they again disappeared.

In cases in which the vertigo is accompanied or has been preceded by gastric derangement, you will often be justified in instituting the treatment for stomachic vertigo; and it will nearly always be successful. However, you must never omit seeking to discover whether the vertigo be not the result of sympathy with some lesion of the liver, kidneys, bladder, or uterus. As you are aware, hepatic and nephritic colics, as well as uterine pains, are often accompanied by nausea, vomiting, and giddiness.

There is another kind of vertigo which greatly resembles stomachic vertigo; viz., that usually consequent upon lesion of the internal ear, and which may be termed *vertigo ab aure læsa*. When speaking of apoplectic cerebral congestion, I mentioned the able communication on this subject which Ménière laid before the Academy of Medicine.

The connection between vertiginous symptoms and diseases of the internal ear were pointed out, in 1863, by Triquet in his clinical lectures;[1] and in a manuscript note communicated to me, he attaches special importance to the vertigo and buzzing in the ear, which occur in cases of inflammation of the labyrinth. He claims for Saissy (de Lyon) the credit of having been the first to point out the coexistence of vertigo with diseases of the ear. I cannot endorse this claim, for in the two cases given in Saissy's work, which appeared in 1827,[2]

[1] Triquet :—Leçons Cliniques sur les Maladies de l'Oreille, p. 113. Paris, 1863.

[2] Saissy :—Essai sur les Maladies de l'Oreille Interne. Lyon, 1827.

cases quoted from the work of Dr. Viricel, he speaks of violent pains depending on lesions of the tympanum, demonstrated by necroscopic examination; but neither in the account given of the cases, nor in Saissy's remarks upon them, is there any mention of vertigo.

It is to Ménière, therefore, that we are indebted for our knowledge of the relation which exists between lesions of the labyrinth and the cerebral symptoms which all physicians prior to him had attributed to the stomach, to apoplectic congestion, or to the commencement of very serious affections of the brain. In 1861, Ménière[1] showed that patients affected with inflammation of the labyrinth present an assemblage of symptoms reputed cerebral, such as vertigo, dizziness, unsteady gait, turning round, and falling—symptoms which are accompanied by nausea, vomiting, and sometimes by syncope.

He became convinced by the frequent observation of cases of this description, that all the symptoms (so far from following the usual course of cerebral affections or stomachic vertigo), disappeared after a period variable in duration, leaving in their place deafness rebellious to treatment, and generally incurable.

Ménière, in the work to which I have alluded, mentions the case of a young woman, who, when menstruating, travelled on a winter night on the outside of a stage-coach, and experienced, as a consequence of this exposure to great cold, *a sudden attack of complete deafness*. When received into Chomel's wards, her principal symptom was constant vertigo: vomiting was induced by the slightest attempt to move; and she died on the fifth day. At the autopsy it was found that the brain, cerebellum, and spinal cord, were free from morbid change. As the patient had all at once become deaf, having had her hearing perfectly good up to the date of the attack, Ménière removed the temporal bones, so that he might be able carefully to examine into the cause of the complete deafness which had supervened so rapidly. The semicircular canals were the only parts of the labyrinth which presented any abnormal appearance: they contained a reddish-coloured plastic lymph, instead of the fluid of Cotugno.

In the cases of Saissy and Triquet, there was found, on necroscopic examination, a similar exudation of a reddish plastic matter, and also thickening of the nervous membrane which lines the semicircular canals. There is, therefore, ground for supposing, that pos-

[1] MÉNIÈRE:—*Bulletin de l'Académie de Médecine*. Vol. XXVI, p. 241.

sibly, had attention been called to the question, there might have been observed during life, symptoms similar to those observed in Ménière's case.

No doubt, Gentlemen, many of you remember the woman, who lay in bed 25 of St. Bernard's ward. She was almost absolutely deaf: whenever she was interrogated in a voice somewhat too loud, her countenance indicated acute suffering, and she complained of great pain in the head, and insupportable noises in the ears, while she was also at the same time seized with vertigo, and held her head between her hands, as if for the purpose of shutting out all external noise. Everything about her, she said, seemed to be turning round. If she were addressed, when standing, in too loud a voice, she would lay hold of the bars of her bed to save herself from falling. She told us, that for a long period, without any appreciable cause, she had suffered from vertigo, which, day by day, was increasing, and had reached such a degree, that she could no longer walk alone in the streets, the noise of the carriages being insupportable, and bringing on the giddiness. She also mentioned that she often felt herself pushed from *left to right;* and that when walking on the pavement, she was always careful to take the right, being afraid of falling upon the causeway. Bear in mind, Gentlemen, that the deafness was greatest on the right side, and that it was on the same side that noise produced a painful impression. The patient frequently had nausea and loss of appetite, although she was not feverish, and had a tongue which did not indicate indigestion. There was no appreciable loss of flesh: she never had had anything wrong in respect of the hepatic and renal secretions, nor the menstrual flux. The cause of the vertiginous symptoms was an affection of the auditory nerve: the nearly constant buzzing in the ear, the almost complete deafness, the exacerbation of the buzzing and pain whenever a noise was made near the patient are facts corroborative of this opinion. On examining the external auditory canal, the membrane of the tympanum was seen to be depressed towards the centre, and to present at that point a sinking down which Triquet attributes to the welding of the ossicula; but this depression of the tympanum, which indicated former inflammation of the middle ear, existed only on the left side, and established a contiguity of pain between the membrana tympani and the fenestra ovalis.

There never occurred in this woman loss of consciousness, convulsions, nor paralysis, and her intellectual faculties remained intact:

it was, therefore, scarcely possible to be satisfied with the hypothesis that there was a lesion of the cerebrum or cerebellum : her sight was good, and she never had strabismus. But on the one hand, on considering the experiments of Flourens, Brown-Séquard, and Vulpian in connection with conclusions arrived at by Ménière, and on the other hand, considering the symptoms of our patient, viz. the deafness, buzzing in the ear, impulse to turn to the right, aad vertigo, it was natural to suppose that the semicircular canals were the seat of a morbid change accounting for all the symptoms we noted. Again, the lesion of the labyrinth, though existing on both sides, was most marked on the right side, the pain was most intense on that side, and the impulse to turn was from *left to right.*

Gentlemen, I cannot upon this occasion set before you in detail the result of each individual experiment of Flourens,[1] results completely confirmed by the researches of Brown-Séquard and Vulpian : I shall only say, that it is now generally admitted by physiologists, that the simultaneous lesion of the semicircular canals of both sides gives rise to forward movement, or backward movement (a tumble head-over-heels), according to the situation of the injured canals; and that when there is lesion on one side only, the propulsion is always to that side. Here, too, pathological anatomy confirms the results of experimental physiology. In 1861, MM. Signol and Vulpian communicated to the *Société de Biologie*[2] the case of a cock which in fighting received a violent stroke on the head from the beak of another cock. The animal was at once stunned, but was soon seen, with head drooping forwards, to turn himself *from left to right,* when attempting to walk. He became blind. When he died, six weeks after receiving the wound, a great part of the right temporal bone was found to be necrosed : the whole of the portion of the bone containing the semicircular canals was isolated by a newly-formed membrane, and it was impossible to discover any trace of the semicircular canals on the right side.

If there really existed in our patient, as I suppose, a lesion of the

[1] Flourens :—Recherches Expérimentales sur les Propriétés et les Fonctions du Système Nerveux. 2de edition (1842), p. 442 et seq.

[2] J. Signol and A. Vulpian :—Note sur un Cas de Nécrose d'une portion du Diploé Cranien chez un Coq. Altération profonde de l'Appareil Auditif : Phénomènes Symptomatiques semblables à ceux que produit la Section des Canaux Semi-circulaires. [Comptes Rendus des Séances de la Société de Biologie, 1861, p. 135. Paris, 1862.]

semicircular canals, the propulsion from left to right would be explained by the morbid change in the canals being greatest on the right side. As for the other symptoms—the vertigo, pain in the head, and nausea—they are sufficiently explained by recollecting that any violent shock imparted to the membrana tympani by a probe or an injection is sufficient to produce buzzing in the ear, vertigo, and nausea. In these cases, the shock is probably transmitted to the fenestra ovalis by the chain of ossicula, and thence to the internal ear, the lesions of which produce the giddiness, as is proved by the case of the young woman reported in Ménière's memoir.

I believe, then, that there was lesion of the semicircular canals in our patient: but if physiology, combined with the clinical and necroscopic study of the cases reported by Ménière, justify our adopting this conclusion in respect of the seat of the lesion, the cause of the lesion remains unexplained. Saissy and Triquet attribute a large share in the etiology to the rheumatic diathesis, and to catarrhal constitutions of the atmosphere: we have seen in the case described by Ménière, how much may depend on exposure to cold and the suppression of the catamenia. In our case, we cannot attribute anything to these causes: because the existence of the rheumatic diathesis was not ascertained, and the patient had not had suppression of the menses.

In conjunction with the case which I have now recapitulated, it will be well to lay two others before you: one of them is detailed by Dr. Burrgraeve,[1] and the other by Dr. Hillairet.[2] In Dr. Burrgraeve's case, there supervened, consequent upon a chill, inflammation of the internal ear, with perforation of the tympanum and discharge of sanguinolent pus from the auditory canal. Upon the sudden suppression of the discharge, the gait became unsteady, the patient being frequently unable to maintain his equilibrium. He soon experienced vertigo, and was obliged to remain close to his bed, to prevent himself from obeying the tendency to turn round. His head was violently drawn from right to left, and from left to right: he felt it impossible to walk, from a feeling that the floor was not firm footing: he felt as if on the deck of a ship rolling in a heavy

[1] Burrgraeve:—*Gazette Médicale de Paris*, 1842, quoted from *Annales et Bulletin de la Société de Médecine de Gand*, 1841.

[2] Hillairet:—Lésions de l'Oreille Interne: Action Réflexe sur le Cervelet et les Pédoncles. [*Comptes Rendus et Mémoires de la Société de Biologie.* Third Series: Vol. III, p. 148, for the year 1861. Paris, 1862.]

sea: he experienced retching and vomiting, having in fact veritable sea-sickness. To quote the patient's own words, as reported by Dr. Burrgraeve:—"When the position of my head was unsettled by my turning it quickly, or by blowing my nose, my legs gave way, and I fell as if struck down by a thunder-bolt." It is a circumstance worthy of attention in this case, that the superior extremities did not participate in the inordination of the inferior, and that they retained precision of movement during the whole course of the disease. The head was perfectly free: there was nothing abnormal in the sight, smell, or taste: nor was there anything morbid in the sense of hearing, except a disagreeable buzzing and singing in the affected ear. The author states that these symptoms in a great measure ceased when the discharge reappeared; but for more than a month afterwards, there remained a certain amount of indecision in the movements.

It is evident that all the symptoms which Ménière has referred to lesions of the labyrinth were present in this case—the vertigo with buzzing in the ear, the nausea, the vomiting, the irresistible tendency to turn round in a determinate sense, the characteristic symptoms, as I have said, of lesions of the semicircular canals.

The case communicated to the *Société de Biologie* by Dr. Hillairet is not less interesting than that described by Dr. Burrgraeve. After a chill, which occasioned acute and long-continued pain in both ears, there supervened a purulent discharge from the right ear. From the time at which this running began, the attacks of pain became less violent, and much less frequent. But the chronic inflammation within the petrous portion of the temporal bone soon manifested itself by numerous fleshy granulations appearing in the external auditory canal in the form of polypus. Subsequently, this polypus, by preventing the pus from escaping, caused the pains to return with their original characters: and in addition, there were buzzing in the affected ear, vertigo, a tendency to vomit, fidgets, feebleness of the inferior extremities, a disposition to stoop, and to turn to the side opposite to that which was the seat of the lesion. Removal of the polypus relieved the patient by giving easy exit to the purulent matter. By-and-by, the discharge gradually dried up under the influence of local and general treatment, whereupon there was an end of the nervous phenomena.

Dr. Hillairet without hesitation connected all these nervous phenomena observed in his patient with a lesion of the semicircular

canals: he then, from physiological experiments and from the case so well analysed by MM. Vulpian and Signol, came to the conclusion that the nervous phenomena were probably the result of lesion of the internal ear.

Grant that all these facts are well established, we shall still have to explain how lesions of the semicircular canals produce disturbance of the brain, that is to say, such symptoms as vertigo, titubation, an irresistible tendency to fall, or to turn round in a determinate direction. We know that all these phenomena may occur when there is lesion of certain parts of the encephalon: but in the cases now under analysis, we must reject the hypothesis of a lesion propagated to the brain by contiguity of the internal ear, inasmuch as the integrity of the cerebral substance is established by the results of clinical study, and by the autopsies made by Ménière, Viricel, and Vulpian.

You know, Gentlemen, the important part now attributed to reflex action in the production of physiological and morbid occurrences: you know that the spinal marrow incited by the sensitive nerve of animal or organic life is a centre of manifold reflex actions: the brain also participates in this action whenever a cranial nerve of general or special sensation is excited in a particular way. Irritation of the ophthalmic branch, without the individual being aware of it, is reflected in a special manner by the brain, and as consequences of this reflection, there occur a flow of tears and injection of the conjunctiva: in the same way, when the retina is excited, there is observed, in virtue of similar reflex action, winking of the eyelids, epiphora, and contraction of the pupils. You are likewise aware of the fact, that the operation for cataract by depression induces vertigo and retching.

Analogous phenomena occur in acute ear-ache. I could multiply examples of this reflex action of the brain. But for the present, let it suffice to remark, that Brown-Séquard in his lectures on the nervous system[1] has announced as his conclusion, that irritation of the auditory nerve, of the optic nerve, and of every sensitive nerve, may, by reflex action, produce convulsions, vertigo, and other symptoms of encephalic disturbance. And, as reflex action may produce its results upon the vaso-motory nervous system, as well as

[1] Brown-Sequard:—Lectures on the Physiology and Pathology of the Central Nervous System, p. 195 et seq. London: 1860.

on the nerves of motion and sensation, it is a legitimate supposition that in lesion of the semicircular canals, reflex action influences the *nervous system of the brain*, in such a way as to produce cerebral anæmia, and consequently many of the symptoms of that form of anæmia, such as giddiness, nausea, and a tendency to syncope.

This last remark brings me to enunciate the hypothesis that stomachic vertigo is perhaps simply the result of a reflex action upon the cerebral circulation, which reflex action has the seat of its incitation in the stomach.

However we may interpret the vertiginous phenomena connected with lesion of the labyrinth or with stomachic derangement, their existence is now quite recognised: you may always refer them to an affection of the organ of hearing, whenever they are accompanied by continuous buzzing in the ear, and followed by deafness.

Gentlemen, it very often happens that stomachic vertigo, particularly when it occurs in the aged, or in those who are verging upon old age, is a prelude to very serious cerebral lesions, such as apoplexy and softening of the brain. Although in the great majority of cases, the vertiginous symptoms which I have described to you leave only a fleeting impression on the brain, and seem to have their origin in the organs of digestion; and although I do not hesitate to employ the treatment which I am going to point out, and which cures the patients—some temporarily (as in the greater number of cases), and others permanently, I am not the less reserved in my statements, knowing that in a few exceptional cases, a simple vertigo is followed by formidable cerebral symptoms too evidently due to very serious encephalic lesion.

To stomachic vertigo assuredly belong the cerebral phenomena which accompany indigestion, gastric discomfort, a simple state of plenitude of the organ, such as occurs after a meal more than usually ample, or after eating some particular articles of food.

These vertiginous symptoms, accompanied by a feeling of weight and dull headache, by ringing and buzzing sounds in the ear, bear a much nearer resemblance than the preceding to the forms of vertigo regarded as depending on cerebral congestion. Even when they are the sole indications of discomfort in the stomach, the circumstances under which they arise, prevent a mistake as to their nature. They are phenomena as transient as the causes which produce them, and they yield with the greatest ease. It is not, indeed, necessary to enlarge on this point, any more than upon the proper treatment.

It is otherwise, however, with the symptoms to which we are now giving our special attention. The ingestion of a small quantity of aliment, of broth, of a little generous wine, or particularly of alcoholic and aromatic liquors, will frequently calm them: but to prevent their return, to cure the disease it is necessary to institute, and perseveringly follow up, a treatment directed against a pathological condition more or less of old standing, and more or less persistent.

There is a plan of treatment which has rendered me great services: I owe my knowledge of it to Bretonneau, my venerated master. You have seen me employ it in our cases, so that you know that it consists in administering bitters and alcalies. The following is the programme.

Every morning, the patient takes an infusion of two grammes of shavings of the wood of quassia amara. The shavings are macerated for twelve hours in a teacupful of cold water. A similar dose may be obtained by allowing the same quantity of water to remain in a goblet made of this bitter wood.

After each of the principal repasts, as well as on going to bed for the night, the patient takes a powder, for which the following is the prescription:—

Bicarbonate of soda . . .	1 gramme.
Prepared chalk . . .	2 grammes.
Magnesia	1 gramme.

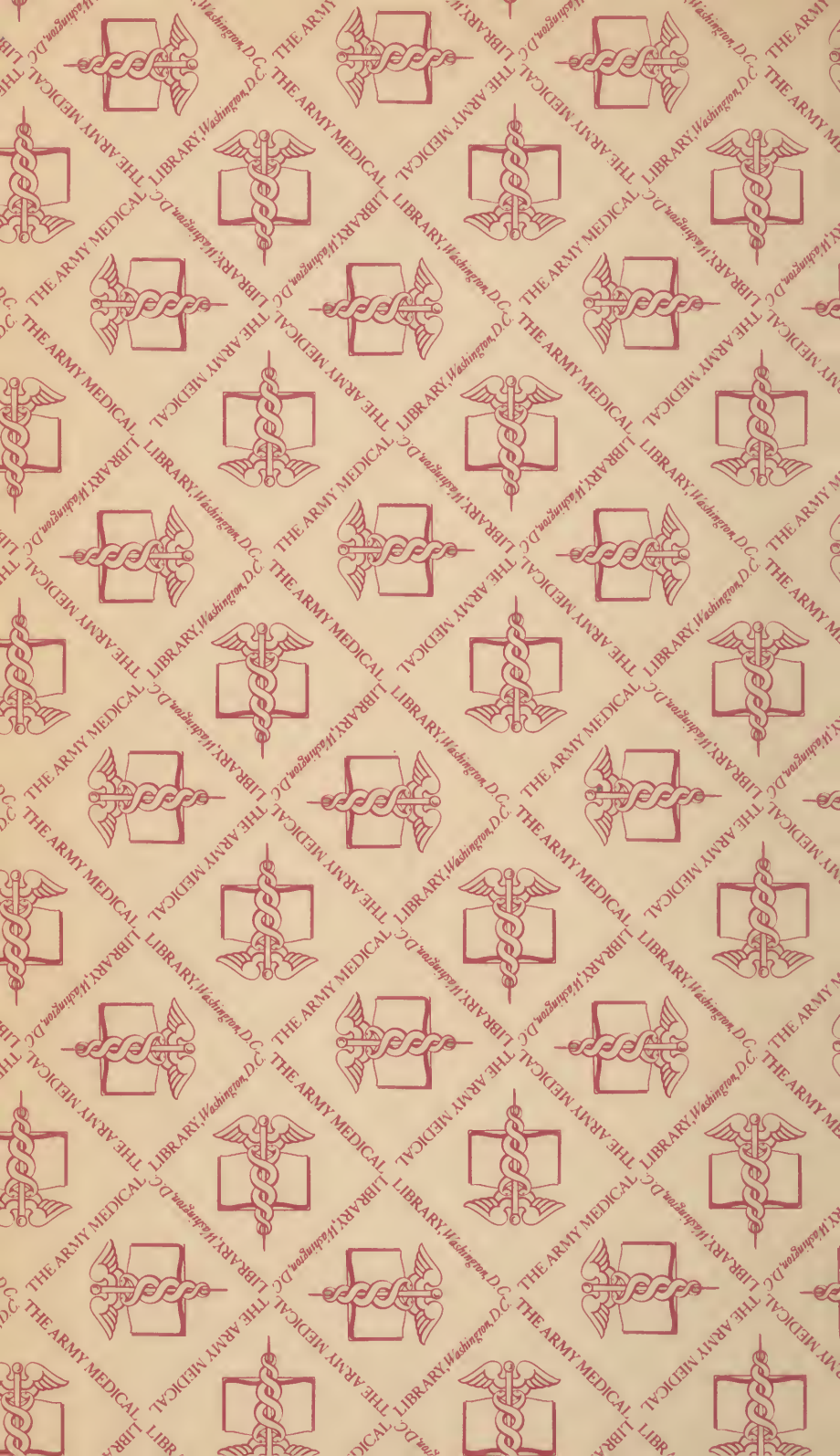
Mix, and divide into three packets, which are to be stirred in half a tumbler of sugared water just before being taken. These alcaline powders ought to be taken for five or six consecutive days, then discontinued, to be resumed after eight or ten days. During the intermission of the powders, the patient ought to drink such natural mineral waters as those of Vichy, Pougues, Vals, or Ems, which seem to act chiefly in virtue of their alcaline ingredients; more than two tumblers for a daily dose must not be taken: or, recourse may be had to the waters of Bussang, Spa, or Schwalbach, which also partly owe their medicinal action to the small quantities of alcalies which they contain, but principally to their ferruginous ingredients.

When the appetite is languid it will be useful to give preparations of the strychnia family of plants to rouse the sensibility of the mucous membrane of the stomach, and the contractility of its mus-

cular coat. These medicines likewise act as excellent tonics. I often prescribe from one to four of the bitter drops of Baumé to be taken at the commencement of meals; or, I order from five to ten drops of the tincture of nux vomica; or the extract of nux vomica in pills, each containing five centigrammes of the extract.

Beyond every other measure, it is essential to insist on a tonic and substantial regimen, assisted by moderate exercise. In a word, the treatment of stomachic vertigo is the treatment of dyspepsia, a subject into which I shall have to enter very fully upon another occasion.

Coltrane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